Resistance to Multiple PRE Herbicides in a Field-evolved Rigid Ryegrass (*Lolium rigidum*) Population

David J. Brunton[1], Peter Boutsalis[2], Gurjeet Gill[3] and Christopher Preston[3]

[1]Postgraduate Student, School of Agriculture Food and Wine, University of Adelaide, Glen Osmond, SA, Australia, [2]Postdoctoral Fellow, School of Agriculture Food and Wine, University of Adelaide, Glen Osmond, SA, Australia and [3]Associate Professor, School of Agriculture Food and Wine, University of Adelaide, Glen Osmond, SA, Australia

Author for correspondence:
David Brunton, School of Agriculture Food and Wine, University of Adelaide, PBM 1, Glen Osmond, SA 5064, Australia. (Email: david.brunton@adelaide.edu.au)

Abstract

A population of rigid ryegrass (*Lolium rigidum* Gaudin) from a field on the Eyre Peninsula, South Australia, was suspected of resistance to thiocarbamate herbicides. Dose–response studies were conducted on this population (EP162) and two susceptible populations (SLR4 and VLR1). The resistant population exhibited cross-resistance to triallate, prosulfocarb, EPTC, and thiobencarb with higher LD_{50} to triallate (14.9-fold), prosulfocarb (9.4-fold), EPTC (9.7-fold), and thiobencarb (13.6-fold) compared with the susceptible populations SLR4 and VLR1. The resistant population also displayed resistance to trifluralin, pyroxasulfone, and propyzamide. The LD_{50} of the resistant population was higher for trifluralin (13.8-fold), pyroxasulfone (8.1-fold), and propyzamide (2.7-fold) compared with the susceptible populations. This study documents the first case of field-evolved resistance to thiocarbamate herbicides in *L. rigidum*.

Introduction

The increase in herbicide-resistant weed species is a major threat to food production globally (Oerke 2006). Long-term use of herbicides as the primary form of weed control has selected for individuals capable of evading herbicide application (Gillespie 2010; Gressel 2009). Currently, 486 monocot and dicot weed species have evolved resistance globally to various herbicide sites of action (Heap 2017). Continued reliance on herbicides for weed control has resulted in new resistance cases reported in Australia and globally, including the evolution of metabolic resistance, which is of great concern, as it can confer unpredictable resistance to various sites of action, including herbicides never used (Beckie et al. 2013; Busi et al. 2013; Yu and Powles 2014).

Multiple weed species currently challenge Australian grain production systems (Llewellyn et al. 2016). The most problematic of these is rigid ryegrass (*Lolium rigidum* Gaudin), which is a genetically diverse, obligate-outcrossing weed species with widespread adaptations across all major cropping regions of southern Australia; it has evolved resistance to multiple herbicides with different sites of action (Busi et al. 2016; Han et al. 2016; Preston and Powles 2002; Yu and Powles 2014). The evolution of resistance in *L. rigidum* to selective POST acetyl-CoA carboxylase (ACCase)-inhibiting (diclofop-methyl) (Heap and Knight 1982) and acetolactate synthase (ALS)-inhibiting (chlorsulfuron) (Powles et al. 1990; Preston and Powles 2002) herbicides, including populations with multiple resistance (Preston et al. 1996), has shifted reliance to PRE herbicides from the thiocarbamate family. Increased reliance on thiocarbamate herbicides has ensued with the evolution of resistance to the PRE dinitroaniline herbicide trifluralin ($3^{(K1)}$: inhibitors of microtubule assembly). Trifluralin resistance is widely documented in southern Australia (Boutsalis et al. 2012; Broster and Pratley 2006). Release of the PRE herbicides prosulfocarb ($8^{(N)}$: inhibitor of fat synthesis) and pyroxasulfone ($15^{(K3)}$: inhibitors of very-long-chain fatty-acid synthesis) provided effective options for the control of *L. rigidum* in cereal and pulse crops (Boutsalis et al. 2014; Tanetani et al. 2013; Walsh et al. 2011). Thiocarbamate herbicides currently provide one of the two effective options for the control of *L. rigidum* in cereals. Resistance evolution to thiocarbamate herbicides threatens the sustainability of Australian grain production. Resistance to the thiocarbamates will reduce the effective control strategies in cereals, thereby restricting the control of *L. rigidum* to one herbicide (pyroxasulfone) (Walsh et al. 2011).

Only a few examples of weeds with resistance to thiocarbamate herbicides exist, one being triallate-resistant wild oat (*Avena fatua* L.) in Canada (Beckie et al. 2004, 2012; O'Donovan et al. 1994) with confirmed cross-resistance to pyroxasulfone and sulfentrazone (Mangin et al. 2016). Resistance to prosulfocarb has also been documented in blackgrass (*Alopecurus*

myosuroides Huds.) populations across Denmark (Keshtkar et al. 2015). In addition, evolution of cross-resistance to prosulfocarb and triallate in *L. rigidum* was achieved by Busi and Powles (2013) in a glasshouse low-dose recurrently selected population MR4 displaying 58% and 35% survival, respectively. Busi and Powles (2016) also demonstrated cross-resistance to prosulfocarb + *S*-metolachlor and pyroxasulfone in *L. rigidum* recurrently selected with either herbicide.

Understanding the levels of resistance to thiocarbamate herbicides is necessary for the development of management strategies to slow the rate of resistance evolution and manage resistant weeds. The evolution of resistance to thiocarbamate herbicides has not yet been fully studied. The objective of this study was to determine the level of resistance and patterns of cross-resistance and multiple resistance in an *L. rigidum* population with suspected field-evolved resistance collected from Cockaleechie, Eyre Peninsula, South Australia, and to test its response to other PRE herbicides.

Materials and Methods

Plant Material

The seed of *L. rigidum* population EP162 (resistant [R]) used in this study was collected from a field in 2014 at Cockaleechie, Eyre Peninsula, South Australia (34.21°S, 135.84°E) with a long history of crop production. Preliminary testing revealed resistance to multiple herbicide sites of action, including ACCase, ALS, microtubule assembly, fat synthesis, and 5-enolpyruvylshikimate-3-phosphate inhibitors. Two well-characterized herbicide-susceptible *L. rigidum* populations, SLR4 and VLR1 (S), were also used in this study (Boutsalis et al. 2012).

Seedling Growth and Herbicide Application

Seeds from EP162, SLR4, and VLR1 were weighed (0.2 g = 50 to 60 seeds) and spread onto the surface of 9.5 cm by 8.5 cm by 9.5 cm punnet pots (Masrac Plastics, SA, Australia) containing cocoa peat potting mix as described by Boutsalis et al. (2012). Herbicides were applied immediately using a laboratory spray cabinet equipped with flat-fan nozzles (Hardi ISO F-110-01, Hardi, Adelaide, SA, Australia) delivering 118 L ha^{-1} water at a pressure of 4 kPa. Control pots were not treated with any herbicide. The experiment was conducted and repeated outdoors under natural growing conditions in winter during the normal growing season (July to August 2017). Pots were watered as needed to maintain potting mix near field capacity. There were three replicates for each herbicide dose, and pots were arranged in a randomized complete block design.

Dose–Response Experiment

Seven PRE herbicides were applied (Table 1) following the method described by Boutsalis et al. (2012). The PRE herbicide triallate was applied to S biotypes at 25, 50, 100, 200, 400, 800, and 1,600 g ha^{-1} and to the R biotype at 400, 800, 1,600, 3,200, 6,400, and 12,800 g ha^{-1}; in Australia, the recommended label rate of triallate for *L. rigidum* control is 1,500 g ha^{-1}. Prosulfocarb was applied to S biotypes at 37.5, 75, 150, 300, 600, 1,200, and 2,400 g ha^{-1} and to the R biotype at 600, 1,200, 2,400, 4,800, 9,600, and 19,600 g ha^{-1}, with the recommended label rate for *L. rigidum* control being 2,400 g ha^{-1}. EPTC was applied to S biotypes at 67.5, 135, 270, 540, 1,080, 2,160, and 4,320 g ha^{-1} and to the R

Table 1. Active ingredients, formulations, and manufacturers of herbicides used in dose–response experiments.

Active ingredient	Trade name	Formulation	Manufacturer
Triallate	Avadex Xtra®	500 g L^{-1}	Nufarm Pty Ltd, Australia
Prosulfocarb	Arcade®	800 g L^{-1}	Syngenta Crop Protection Pty Ltd, Australia
EPTC	Eptam®	720 g L^{-1}	Crop Care Australasia Pty Ltd, Australia
Thiobencarb	Saturn®	800 g L^{-1}	Bayer Crop Science Pty Ltd, Australia
Trifluralin	Trifluralin	480 g L^{-1}	Imtrade Australia Pty Ltd, Australia
Propyzamide	Kerb®	500 g L^{-1}	Dow AgroSciences Pty Ltd, Australia
Pyroxasulfone	Sakura®	850 g kg^{-1}	Bayer Crop Science Pty Ltd, Australia

biotype at 1,080, 2,160, 4,320, 8,640, 17,280, and 34,560 g ha^{-1}, with the recommended label rate for *L. rigidum* control being 4,320 g ha^{-1}. Thiobencarb is not a registered herbicide for the control of *L. rigidum* in Australia but was included as another thiocarbamate herbicide and applied to S biotypes at 56.25, 112.5, 225, 450, 900, 1,800, and 3,600 g ha^{-1} and to the R biotype at 900, 1,800, 3,600, 7,200, 14,400, and 28,800 g ha^{-1}.

Additional PRE herbicides with different sites of action were included, as these provide an alternative option for the control of *L. rigidum* in Australia. Trifluralin was applied to S biotypes at 7.81, 15.62, 31.25, 62.5, 125, 250, and 500 g ha^{-1} and to the R biotype at 125, 250, 500, 1,000, 2,000, and 4,000 g ha^{-1}, with the recommended label rate for *L. rigidum* control being 500 g ha^{-1}. Propyzamide was applied to S biotypes at 7.81, 15.62, 31.25, 62.5, 125, 250, and 500 g ha^{-1} and to the R biotype at 125, 250, 500, 1,000, 2,000, and 4,000 g ha^{-1}, with the recommended label rate for *L. rigidum* control being 500 g ha^{-1}. Pyroxasulfone was applied to S biotypes at 1.6, 3.2, 6.4, 12.8, 25.5, 51, and 102 g ha^{-1} and to the R biotype at 25.5, 51, 102, 204, 408, and 816 g ha^{-1}, with the recommended label rate for *L. rigidum* control being 100 g ha^{-1}. Pots were assessed at 28 d after herbicide treatment, and plants that had emerged and grown to the 2-leaf stage were counted. Emergence was assessed as the number of plants germinating in the treated pots compared with the average number germinating in control pots and calculated as a percentage.

Statistical Analysis

Following an ANOVA, both experiment runs were pooled (no statistical difference between experimental runs) and analyzed using a log-logistic equation (GraphPad Prism v. 7.0, GraphPad Software, San Diego, CA) fit to the survival data. LD$_{50}$ (dose required to reduce emergence by 50% of individuals in the population) parameter estimates from the log-logistic analysis were used to calculate the resistance index (RI), which is the resistant/susceptible ratio of the LD$_{50}$. The model fit was

$$y = \frac{100}{1 + 10^{(\log \text{IC}_{50} - x) \times b}} \qquad [1]$$

where *y* represents plant survival (%) or biomass reduction (%), *x* is the log-dose of the herbicide used, IC$_{50}$ is the herbicide dose
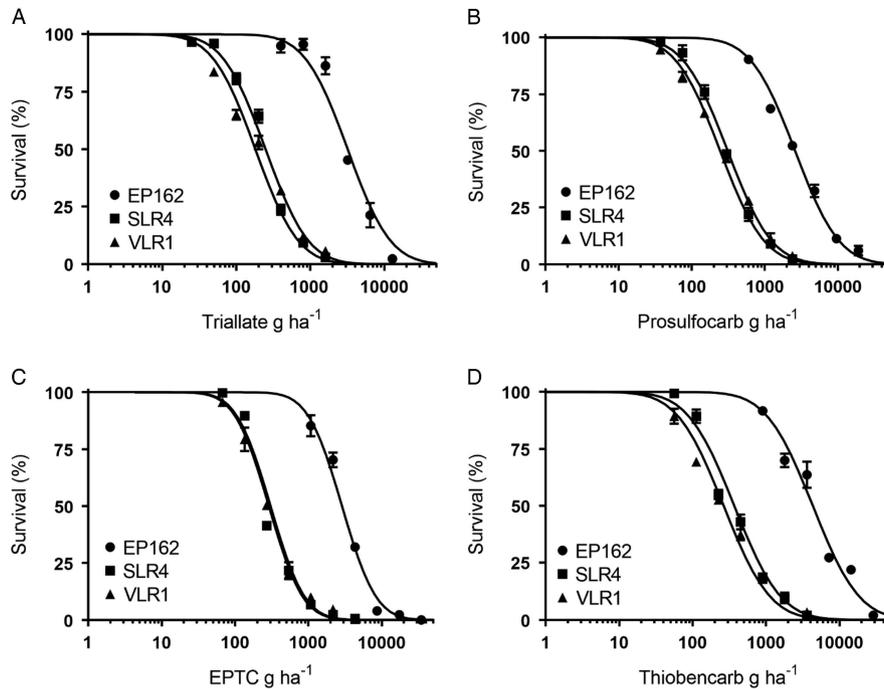
Figure 1. Response of EP162, SLR4, and VLR1 populations of *Lolium rigidum* treated with varying rates of triallate (A), prosulfocarb (B), EPTC (C), and thiobencarb (D). Each data point is the mean of six replicates, and bars represent standard error of the mean.

required to cause 50% reduction in plant emergence (LD_{50}), and *b* denotes the slope of the curve.

Results and Discussion

Plants of the S populations SLR4 and VLR1 were completely controlled at $1,500 \, g \, ha^{-1}$ triallate, the recommended field rate, while the resistant population EP162 showed no mortality at this rate (Figure 1A). The triallate rates required for 50% mortality (LD_{50}) for the SLR4 and VLR1 populations were $248 \, g \, ha^{-1}$ and $181 \, g \, ha^{-1}$, respectively (Table 2). The LD_{50} for the resistant population EP162 was $3,188 \, g \, ha^{-1}$, giving an RI 14.9-fold higher than the mean of both S populations. Prosulfocarb at the recommended rate of $2,400 \, g \, ha^{-1}$ controlled both S populations, while EP162 had 48% mortality at this rate (Figure 1B). The LD_{50} rates for SLR4 and VLR1 were $311 \, g \, ha^{-1}$ and $246 \, g \, ha^{-1}$, respectively, and $2,608 \, g \, ha^{-1}$ for EP162 (9.4-hold higher than the S populations). EPTC completely controlled both S populations at the recommended rate of $4,320 \, g \, ha^{-1}$, while the EP162 population showed 68% mortality at this rate (Figure 1C). The LD_{50} rates for the SLR4 and VLR1 populations were $305 \, g \, ha^{-1}$ and $288 \, g \, ha^{-1}$, while the LD_{50} for the EP162 population was $2,867 \, g \, ha^{-1}$ (9.7-fold higher than the S populations). Even though thiobencarb is not registered for *L. rigidum* control in Australia, it completely controlled both S populations at $3,600 \, g \, ha^{-1}$ (Figure 1D). The LD_{50} rates for thiobencarb for the SLR4 and VLR1 populations were $370 \, g \, ha^{-1}$ and $265 \, g \, ha^{-1}$, respectively, and $4,332 \, g \, ha^{-1}$ for the EP162 population (13.6-fold higher than the S populations).

Trifluralin completely controlled the SLR4 and VLR1 populations at the recommended rate of $500 \, g \, ha^{-1}$, with the EP162 population recording 45% mortality at this rate (Figure 2A). The LD_{50} rates for SLR4 and VLR1 were $39 \, g \, ha^{-1}$ and $27 \, g \, ha^{-1}$, respectively, and $455 \, g \, ha^{-1}$ for EP162 (13.8-fold higher than the S populations) (Table 2). Propyzamide completely controlled

both the S and R populations at the recommended rate of $500 \, g \, ha^{-1}$ (Figure 2B). The LD_{50} rates for the SLR4 and VLR1 populations were $30 \, g \, ha^{-1}$ and $23 \, g \, ha^{-1}$, respectively, and $74 \, g \, ha^{-1}$ for the EP162 population (2.7-fold greater than the S populations). Pyroxasulfone completely controlled both S populations at the recommended rate of $100 \, g \, ha^{-1}$. The LD_{50} rates for the SLR4 and VLR1 populations were $9.5 \, g \, ha^{-1}$ and $6.2 \, g \, ha^{-1}$, respectively. The EP162 population displayed 28% survival to pyroxasulfone at the recommended rate (Figure 2C) and an LD_{50} of $64 \, g \, ha^{-1}$ (8.1-fold greater than the S populations).

The EP162 population of *L. rigidum* showed cross-resistance and a similar RI to the thiocarbamate herbicides triallate, prosulfocarb, EPTC, and thiobencarb (Table 2). This suggests a similar resistance mechanism may be providing resistance to the thiocarbamate herbicides in this population. Resistance to triallate

Table 2. Pooled dose–response data of triallate, prosulfocarb, EPTC, thiobencarb, trifluralin, propyzamide, and pyroxasulfone required for 50% mortality (LD_{50}) of resistant and susceptible *Lolium rigidum* populations with 95% confidence intervals (in parentheses) and resistance index (RI).

Population	SLR4	VLR1	EP162	RI[a]	P[b]
		—LD_{50} in g ai ha^{-1}—			
Triallate	248 (201, 308)	181 (151, 217)	3,188 (2,347, 4,347)	14.9	*
Prosulfocarb	311 (259, 375)	246 (209, 289)	2,608 (2,012, 3,374)	9.4	*
EPTC	305 (238, 390)	288 (231, 357)	2,867 (2,005, 4,070)	9.7	*
Thiobencarb	370 (283, 482)	265 (207, 335)	4,332 (2,963, 6,317)	13.6	*
Trifluralin	39 (32, 46)	27 (23, 32)	455 (353, 586)	13.8	*
Propyzamide	30 (26, 34)	23 (20, 26)	74 (45, 97)	2.7	*
Pyroxasulfone	9.5 (7.6, 11.3)	6.2 (5.1, 7.6)	64 (46, 88)	8.1	*

[a]RI as compared with the standard susceptible populations (SLR4 and VLR1).
[b]Probability value indicates significant differences in LD_{50} values between *L. rigidum* populations treated with a particular herbicide.
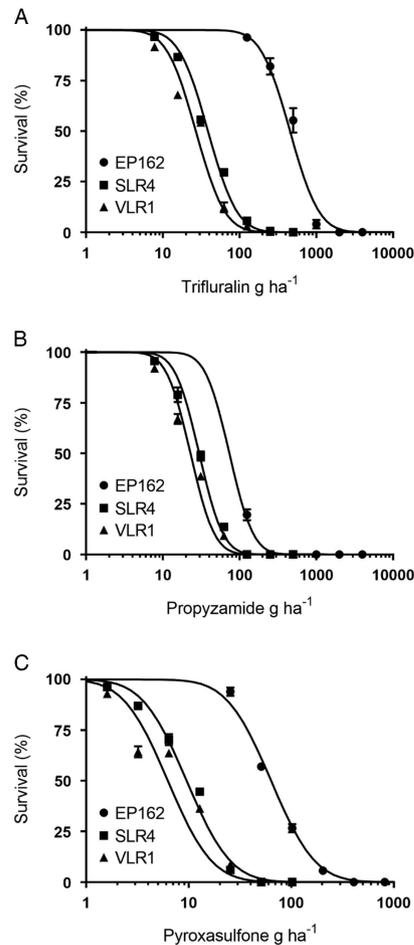*$P < 0.01$.

Figure 2. Response of EP162, SLR4, and VLR1 populations of *Lolium rigidum* treated with varying rates of trifluralin (A), propyzamide (B), and pyroxasulfone (C). Each data point is the mean of six replicates, and bars represent standard error of the mean.

in *A. fatua* has previously been reported by Kern et al. (1996) to occur through reduced herbicide activation (sulfoxidation), and such a mechanism may provide cross-resistance to other thiocarbamate herbicides. However, reduced activation does not explain resistance in this population to other PRE herbicides, such as trifluralin, that do not require sulfoxidation for activation (Kern et al. 1996). The EP162 population shows a response profile more similar to an *A. myosuroides* population from Denmark as reported by Keshtkar et al. (2015), which has a non–target site resistance mechanism conferring strong resistance to the herbicides prosulfocarb and pendimethalin.

The level of resistance to prosulfocarb observed in this field-selected population was similar to a recurrently selected *L. rigidum* population of Busi and Powles (2013). The RIs for triallate and thiobencarb were slightly higher than for prosulfocarb and EPTC, which could be related to the differences in water solubility and binding capacity among the thiocarbamate herbicides tested (Mangin et al. 2017; Westra et al. 2014). The EP162 population displayed a level of trifluralin resistance similar to a recent report of 17-fold resistance conferred through a target-site point mutation in an *L. rigidum* population SLR74 (Fleet et al. 2017) and consistent with reported resistance in field-collected populations from across South Australia (Boutsalis et al. 2012). This observed level of resistance to other herbicide sites of action may be the

result of enhanced herbicide metabolism via cytochrome P450 enzymes (Busi et al. 2017a) or by glutathione *S*-transferase conjugation with pyroxasulfone as reported by Busi et al. (2017b) in *L. rigidum*.

Dose–response experiments also identified resistance to pyroxasulfone. Despite this, pyroxasulfone still provided a considerable level of control (72%) at the recommended field rate of 100 g ha^{-1} (Figure 2C). In contrast, the resistance observed to propyzamide (3$^{(K1)}$: inhibitors of microtubule assembly) was much lower than pyroxasulfone. This herbicide still provided effective control of the population at the recommended rate. Even though propyzamide has the same site of action as trifluralin, propyzamide inhibits polymerization of tubulin into microtubules via a different mechanism compared with trifluralin as reported by Hoffman and Vaughn (1994). These differences in resistance can be explained by the repeated use of trifluralin leading to higher levels of resistance compared which propyzamide, which has been used primarily in canola (*Brassica napus* L.) after being registered in Australia in 2014. Variations in resistance within a site of action are known; different target-site mutations can result in different patterns of resistance to herbicides within a site of action (Preston 2017). In addition, herbicides belonging to the same site of action but to different chemical families will bind differently. One herbicide may be unable to bind, leading to resistance, while the binding of others may not be affected, leading to greater susceptibility (Oettmeier et al. 1991).

This study is the first documented case of field-evolved resistance to thiocarbamate herbicides in a population of *L. rigidum*. In response to widespread trifluralin resistance, use of the herbicide prosulfocarb has allowed growers to control *L. rigidum* in wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L.) in Australia. However, resistance evolution to thiocarbamate herbicides will result in increased reliance by growers on alternative herbicides such as pyroxasulfone and propyzamide. Potential resistance evolution to pyroxasulfone is of great concern, as this herbicide currently provides the most effective way of managing multiply resistant *L. rigidum* populations in wheat. The resistance to pyroxasulfone detected in the EP162 population suggests resistance to this herbicide could occur due to selection with trifluralin and/or prosulfocarb as reported by Busi and Powles (2016). Control of *L. rigidum* in canola is currently achieved with propyzamide (3$^{(K1)}$); however, overreliance on propyzamide also increases the risk of resistance evolution to this herbicide. Evolution of cross-resistance and multiple resistance to PRE herbicides has made *L. rigidum* management increasingly difficult in Australian cropping systems. Australian growers will need to consider alternative strategies in addition to using propyzamide and pyroxasulfone to effectively manage *L. rigidum* in the future. These strategies will need to include mixtures of PRE herbicides, increasing crop competition with weeds, and the adoption of strategies to reduce weed seeds returning to fields (Beckie 2006; Walsh and Powles 2007).

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