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ACCase-Inhibiting Herbicide-Resistant *Avena* spp. Populations from the Western Australian Grain Belt

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Avena spp. are world weeds with many cases of evolved herbicide resistance. In Australia, *Avena* spp. (wild oat and sterile oat) are a major problem, especially in grain crops. Acetyl-CoA carboxylase (ACCase)–inhibiting herbicides have been used extensively since the late 1970s for *Avena* spp. control. However, continued reliance on these herbicides has resulted in the evolution of resistant *Avena* spp. populations. Resistance across many ACCase-inhibiting herbicides was characterized in four *Avena* spp. populations from the Western Australian grain belt. Dose–response experiments were conducted to determine the level of resistance to the aryloxyphenoxypropionates and cyclohexanediones and to the phenylpyrazoline herbicide pinoxaden. On the basis of resistance index values, all four resistant populations exhibited high-level diclofop resistance but varied in the level of resistance to other ACCase-inhibiting herbicides tested. It is evident that *Avena* spp. populations from the Western Australian grain belt have evolved resistance to a number of ACCase-inhibiting herbicides.
Nomenclature: Diclofop; pinoxaden; sterile oat, *Avena sterilis* L. AVEST; wild oat, *Avena fatua* L. AVEFA.
Key words: Herbicide resistance, cross-resistance patterns.

Avena spp. son malezas mundiales con muchos casos de evolución de resistencia a herbicidas. En Australia, *Avena spp.*, (*A. fatua* y *A. sterilis*), representan un gran problema, especialmente en cultivos de grano. Herbicidas inhibidores de la acetil-CoA carboxylase (ACCase) se han usado extensivamente desde finales de los años 1970's para el control de *Avena spp.* Sin embargo, la continua dependencia en estos herbicidas ha resultado en la evolución de poblaciones de *Avena spp.* resistentes. La resistencia a varios herbicidas inhibidores ACCase fue caracterizada en cuatro poblaciones de estas malezas en el cinturón de granos del occidente de Australia. Se realizaron experimentos de respuesta a dosis para determinar el nivel de resistencia a los aryloxyphenoxypropionates (APPs), a los cyclohexanediones (CHDs), y al pinoxaden, un herbicida phenylpyrazoline (PPZ). Basado en el índice de valores de resistencia, todas las cuatro poblaciones resistentes exhibieron altos niveles de resistencia al diclofop, pero variaron en el nivel de resistencia a otros herbicidas inhibidores ACCase. Es evidente que las poblaciones de *Avena spp.* del cinturón de granos del occidente australiano han desarrollado resistencia a un número de herbicidas inhibidores ACCase.

Acetyl-CoA carboxylase (ACCase), the key enzyme involved in the first step of fatty acid biosynthesis in plants, is the primary target of ACCase-inhibiting herbicides (reviewed by Délye 2005; Devine and Shimabukuro 1994). These herbicides comprise three chemically distinct herbicide classes: the aryloxyphenoxypropionates (APPs), cyclohexanediones (CHDs), and the more recent phenylpyrazoline (PPZ), with a single herbicide pinoxaden. The mode of action of all these herbicides is inhibition of the homomeric plastidic ACCase in nearly all grass species. As a consequence of ACCase inhibition, fatty acid biosynthesis is halted, resulting in plant death in grass species. However, in the great majority of dicotyledonous species, their heteromeric chloroplastic ACCase is insensitive to the APP, CHD, and PPZ herbicides (reviewed by Délye 2005; Devine 1997; Devine and Shimabukuro 1994; Powles and Yu 2010). Thus, nearly all dicotyledonous species exhibit target-site insensitivity to these herbicides. Importantly, cereal crop species such as wheat (*Triticum aestivum* L.), corn (*Zea mays* L.), rice (*Oryza sativa* L.), and barley (*Hordeum vulgare* L.) are tolerant to certain ACCase-inhibiting herbicides because they can rapidly metabolize the herbicides to nontoxic forms. This is the basis of selectivity for some ACCase-inhibiting herbicides in cereal crops (Devine 1997).

Since the introduction of the first APP herbicide, diclofop, in 1974 (Miller and Nalewaja 1974) and the first CHD herbicide, sethoxydim, in 1983 (Devine and Shimabukuro 1994), many ACCase-inhibiting herbicides have been commercialized and used persistently and globally to control many grass weed species. However, the practice of persistent use of one herbicide mode of action is a potent selection for the evolution of resistant weed biotypes. From the first reports (Heap and Knight 1982; Moss and Cussans 1985), many grass weed species have evolved resistance to ACCase-inhibiting herbicides. Currently, resistance to ACCase-inhibiting herbicides has been documented in 41 grass species from 31 countries (Heap 2011), with most ACCase-inhibitor resistance cases associated with ryegrass (*Lolium* spp.), blackgrass (*Alopecurus myosuroides* Huds.), and wild oats (*Avena* spp.) (reviewed by Délye 2005; Devine and Shimabukuro 1994; Powles and Yu 2010).

Avena spp. are one of the world's major grass weed problems in cropping areas and are the second most important herbicide-resistant weed species worldwide (Heap 2011). *Avena* spp. mainly infest temperate zone crops, and

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Table 1. Frequency of resistant individuals of selected resistant (R1, R2, R3, and R4) *Avena* spp. populations to each acetyl-CoA carboxylase–inhibiting herbicide screened at upper recommended field rates. Table was modified from Owen and Powles (2009).

			Survival			
Chemical class	Herbicide	Rate	R1	R2	R3	R4
		g ha^{-1}	%			
Aryloxyphenoxy-propionate	Diclofop	563	90	96	100	94
	Fenoxaprop-P	38.5	59	73	100	76
	Clodinafop	18	62	78	74	80
Cyclohexanedione	Clethodim	60	0	4	20	12
	Sethoxydim	186	84	69	100	63
	Tralkoxydim	200	2	4	0	14
Phenylpyrazoline	Pinoxaden	20	8	33	12	20

ACCase-inhibiting herbicides are widely and regularly used for control. Consequently, resistance to ACCase-inhibiting herbicides has evolved in many crop field *Avena* spp. populations in Australia (Boutsalis 2007; Liu et al. 2007; Maneechote et al. 1994, 1997; Mansooji et al. 1992; Owen and Powles 2009; Widderick and Cook 2011), North America (Beckie et al. 1999; Heap et al. 1993; Seefeldt et al. 1994; Shukla et al. 1997), Turkey (Uludag et al. 2007), and in other parts of the world (Heap 2011).

A large random survey conducted in 2005 across the large Western Australian (WA) grain belt found that 71% of the 150 *Avena* spp. (wild oat or sterile oat) populations tested were resistant to diclofop (Owen and Powles 2009). Only some of these populations displayed resistance across other APP, CHD, and PPZ herbicides. In particular, four populations survived many ACCase-inhibiting herbicides. However, while resistance to ACCase-inhibiting herbicides in *Avena* spp. populations is well known in other cropping regions of Australia, few studies have quantified the whole-plant dose–response and cross-resistance patterns of resistant *Avena* spp. populations from the WA grain belt. The present study quantifies resistance across ACCase-inhibiting herbicides in these four chosen resistant *Avena* spp. populations from the WA grain belt. This information is important for developing possible management strategies for the control of these resistant populations.

Materials and Methods

Seed Collection and Plant Material. Seeds from *Avena* spp. populations were collected from 150 cropping fields in a large random survey in 2005. In May to September 2006 and 2007, collected seeds were germinated and screened with commercial herbicides (including ACCase-inhibiting herbicides). Seed collection and herbicides screening are detailed in Owen and Powles (2009). From the initial herbicide screening, four resistant *Avena* spp. populations (three wild oat populations M3/4, M3/5, M3/27, and one sterile oat population, M5W/7, hereafter referred to as R1, R2, R3, R4, respectively) with resistance to several ACCase-inhibiting herbicides were chosen for this study (Table 1). Seeds of a herbicide-susceptible wild oat population (hereafter referred to as S population) were collected in December 2005 from the South Australian Flinders Range (31°25′52.3″S, 138°43′40.1″E) in an area hundreds of kilometers from cropping regions and with no known herbicide exposure. For the four resistant populations during the survey screening, plants surviving herbicide application were grown for seed increase in 2007. During the 2008 growing season, seeds from these resistant populations and the S population were again grown for further seed increase under the same growing conditions to obtain enough seeds for subsequent herbicide evaluation (this study). All bulked seeds were stored in a non–air conditioned glasshouse until use.

Whole-Plant Herbicide Dose Response. All experiments were conducted with potted plants maintained in the field during the normal growing season (May–September 2009) at the University of Western Australia. In mid-April 2009, approximately 800 seeds from each of the four resistant populations plus the S population were germinated in 500-ml plastic containers containing 0.6% agar solidified water. To improve germination, the seed's embryo from all resistant populations was lightly indented with two-tooth tweezers before placing on the 0.6% agar solidified water. The containers were stored at 4 to 5 C for 10 d until the seeds began to germinate then were transferred to room temperature (20 to 25 C) for 3 d. Twenty seedlings of 3-cm height from each population were transplanted at a depth of 1 cm in 180-mm-diam plastic pots filled with potting mix (50% composted pine bark, 25% peat, 25% river sand; the University of Western Australia, Crawley, Australia). Plants were grown outdoors, watered, and fertilized regularly. At the three- to four-leaf stage, seedlings were sprayed with selected ACCase-inhibiting herbicides.

Commercial herbicide formulations were used in all studies. ACCase-inhibiting herbicides (diclofop [Hoegrass® 500], fenoxaprop-P [Wildcat®], and sethoxydim [Sertin®], Bayer CropScience, East Hawthorn, Australia; clethodim [Select®], Sumitomo Chemical Australia, Chatswood, Australia; and pinoxaden [Axial®], Syngenta Crop Protection Pty. Ltd., Level 1, Macquarie Park, Australia) were applied using a custom-built, dual nozzle (TeeJet® XR11001 flat fan, TeeJet Australasia Pty Ltd, Newtown, Australia) cabinet sprayer delivering herbicide in 112 L ha^{-1} water at 210 kPa, at a speed of 3.6 km h^{-1}. For resistant populations, herbicides were applied at the following rates: diclofop at 0, 138, 275, 550, 1,100, 2,200, 4,400, and 8,800 g ha^{-1}; fenoxaprop-P at 0, 9.63, 19.2, 38.5, 77, 154, and 308 g ha^{-1}; clethodim at 0, 6, 15, 30, 60, and 120 g ha^{-1}; sethoxydim at 0, 46.5, 93, 186, 372, 744, and 1,490 g ha^{-1}; and pinoxaden at 0, 5, 10, 20, 40, 80, and 160 g ha^{-1}. The S population was treated with diclofop at 0, 138, 275, 550, 1,100, 2,200, and 4,400 g ha^{-1}; fenoxaprop-P at 0, 0.963, 1.92, 3.85, 9.63, 19.2, 38.5, and 77 g ha^{-1}; clethodim at 0, 0.75, 1.5, 3, 6, 15, 30, and 60 g ha^{-1}; sethoxydim at 0, 4.65, 9.3, 18.6, 46.5, 93, 186, and 372 g ha^{-1}; and pinoxaden at 0, 0.5, 1, 2.5, 5, 10, 20, and 40 g ha^{-1}.

To enhance herbicide performance, BS1000 surfactant (Crop Care Australasia Pty. Ltd., Murarrie, Australia) was added to diclofop, fenoxaprop-P, and sethoxydim; Hasten™ spray adjuvant (Victorian Chemical Co. Pty. Ltd., Coolaroo, Australia) was added to clethodim; and Adigor® spray

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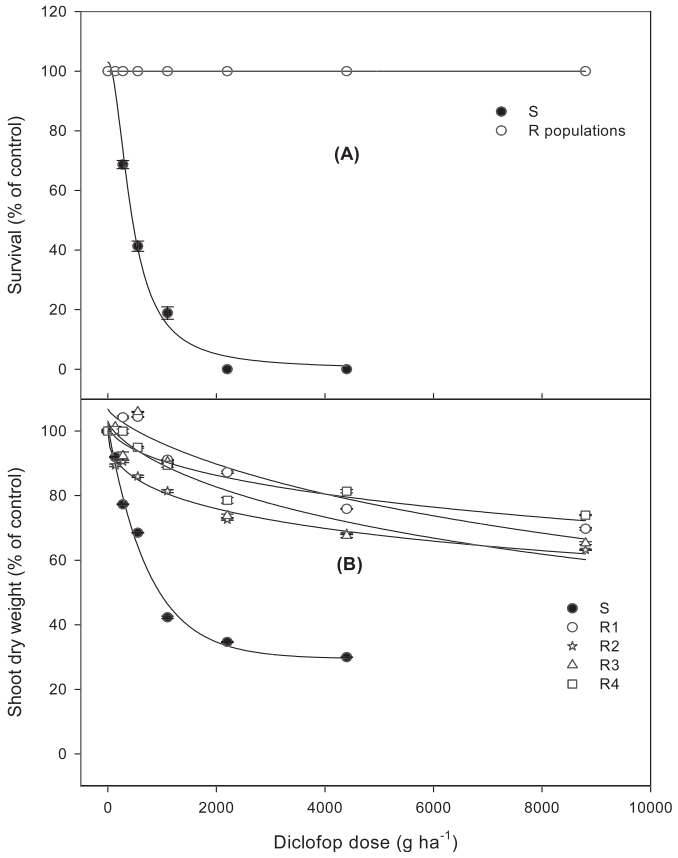

Figure 1. Survival (A) and shoot dry weight (B) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations to diclofop 21 d after treatment. Registered or recommended rate for diclofop is 550 g ha^{-1}. Bars indicate the standard errors of the means of the three replicates.

adjuvant (Syngenta Crop Protection Pty. Ltd., Level 1, Macquarie Park, Australia) was added to pinoxaden. All herbicides were sprayed in mid-May 2009. The average daytime temperatures during the experiment ranged from 18 to 21 C. Visual assessments of plant survival were made 21 d after herbicide treatment. Plants were considered resistant if they survived and continued to produce new growth after herbicide treatment and susceptible if they displayed severe symptoms of leaf chlorosis, desiccation, retarded growth, or no new active growth and eventually plant death, similar to the S population. Plants were harvested 1 cm aboveground, dried at 65 C for 72 h, and weighed. The mean dry weight of all plants (dead and alive) was calculated for each population and expressed as a percentage of the untreated controls for that population.

Statistical Analysis. All herbicide experiments were arranged in a randomized complete block design with three replications per treatment. Nonlinear regression (Sigmaplot® version 11 SPSS Inc., Build 11.0.0.77 Copyright© 2008, Systat Software Inc., GmbH Schimmelbuschstrasse, Germany) was used to generate herbicide dose–response curves. The herbicide rate resulting in 50% mortality (LD_{50}) and the herbicide rate required to reduce mean dry weight by 50% (GR_{50}) were calculated using the logistic model (Equation 1):

$$y = a/1 + (x/I_{50})^{b} \quad [1]$$

where y is the plant survival or shoot dry weight expressed as a percentage of control at herbicide rate = x, a is the maximum plant survival or shoot dry weight attained (%), I_{50} is the dose causing 50% response (LD_{50} for survival data or GR_{50} for shoot dry weight data), and b is the slope around LD_{50} or GR_{50}.

For data that did not fit the above logistic model, a three-parameter exponential decay model with a lower asymptote was used (Equation 2):

$$y = y_0 + ae^{-bx} \quad [2]$$

where y_0 is the lower limit, $a + y_0$ is the upper limit, b is the slope, and x is the dose causing 50% response. For the data that did not fit the above equations (where reduction in the survival or shoot dry weight was less than 50%), the LD_{50} and GR_{50} values were indicated as greater (>) than the highest rate used for each herbicide. Resistance index (R/S) for survival data was calculated as the LD_{50} value of the resistant population divided by the LD_{50} value of the S population. A similar R/S index calculation was used for GR_{50} values.

Results and Discussion

Resistance level to the APP, CHD, and PPZ herbicides for the resistant populations have been classified as high (> 12), moderate (> 6 to 12), low (2 to 6), and sensitive (< 2) according to the R/S index of LD_{50} and GR_{50} values. All four *Avena* spp. populations were found to exhibit high-level resistance to diclofop. No S plants survived 2,200 g ha^{-1}, whereas 100% survival was observed for each resistant population, even at the highest rate of 8,800 g ha^{-1} (Figure 1A). The diclofop LD_{50} for the S population was 441 g ha^{-1}, whereas LD_{50} values greater than 8,800 g ha^{-1} were recorded in all resistant populations, giving resistance index (R/S) values more than 19-fold (Table 2). For all four resistant populations, diclofop only slightly reduced shoot dry weight (Figure 1B), with the R/S values for all resistant populations estimated at more than ninefold (Table 3).

Cross-resistance to other ACCase-inhibiting herbicides varied among populations. For the APP herbicide fenoxaprop-P, population R1 exhibited moderate-level resistance, whereas R2, R3, and R4 showed high-level resistance (Figure 2A). The LD_{50} value for population R1 was eightfold greater than the S population, whereas LD_{50} values greater than 15-fold were observed in populations R2, R3, and R4 (Table 2). Similar to diclofop, fenoxaprop-P did not substantially reduce the shoot dry weight of any of the four resistant populations at the field rate (38.5 g ha^{-1}; Figure 2B). The lowest fenoxaprop-P GR_{50} value for resistant populations was observed in population R1 (3.5-fold greater than the S population), whereas a GR_{50} value greater than 25-fold was recorded in population R4 (Table 3).

For the CHD herbicide sethoxydim, population R1 was found to exhibit low-level resistance, whereas the other three populations (R2, R3, and R4) exhibited moderate-level resistance (Figure 3A). The sethoxydim LD_{50} values for

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Table 2. Parameters of the log-logistic analysis of acetyl-CoA carboxylase–inhibiting herbicide dose required to cause 50% mortality (LD_{50}) and resistance index (R/S) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations. Standard errors are in parentheses.

Population	Herbicide	a	b	LD_{50}	r^2 coefficient	R/S ratio
				g ha^{-1}		
S	Diclofop	103 (4.9)	1.9 (0.3)	441 (44.8)	0.99	
R1		—	—	> 8,800	1.0	> 19
R2		—	—	> 8,800	1.0	> 19
R3		—	—	> 8,800	1.0	> 19
R4		—	—	> 8,800	1.0	> 19
S	Fenoxaprop-P	100.9 (1.9)	2.2 (0.2)	20 (1.1)	0.99	
R1		100.7 (4.2)	1.8 (0.4)	161 (20)	0.97	8
R2		100.9 (0.9)	1.4 (0.1)	> 308	0.99	> 15
R3		102 (4.1)	1.4 (0.5)	> 308	0.90	> 15
R4		101.1 (2.2)	1.1 (0.5)	> 308	0.90	> 15
S	Sethoxydim	—	—	96[a]	0.92	
R1		102.4 (3.2)	2.4 (0.3)	281 (19.8)	0.99	3
R2		100.2 (1.6)	1.6 (0.1)	1,023 (52.1)	0.99	10.5
R3		98.2 (2.2)	3.4 (0.6)	699 (35)	0.99	7
R4		101.2 (1.8)	2.4 (0.3)	1,012 (52)	0.99	10.5
S	Clethodim	98.7 (0.8)	6.9 (0.9)	3.5 (0.1)	0.99	
R1		100 (0.1)	3.6 (0.1)	9 (0.2)	1.0	2.6
R2		102.7 (5.2)	3.1 (0.7)	12 (1.1)	0.99	3.4
R3		100 (0.3)	5 (0.1)	11 (0.1)	1.0	3
R4		101.1 (1.3)	2.6 (0.1)	23 (0.6)	0.99	6.6
S	Pinoxaden	100.2 (0.4)	4.1 (0.1)	3.4 (0.03)	0.99	
R1		100.4 (0.7)	2.3 (0.1)	18.6 (0.3)	0.99	5.5
R2		103.7 (6.3)	1.4 (0.2)	24.8 (4.2)	0.98	7
R3		102 (3)	3.1 (0.4)	12 (0.6)	0.99	3.5
R4		103.2 (5.7)	1.1 (0.2)	69 (12.8)	0.96	20

[a] The LD_{50} value was calculated from parameters obtained from exponential decay (Equation 2).

Table 3. Parameters of the log-logistic analysis of acetyl-CoA carboxylase–inhibiting herbicide dose required to reduce shoot dry weight (GR_{50}) by 50% and resistance index (R/S) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations. Standard errors are in parentheses.

Population	Herbicide	a	b	GR_{50}	r^2 coefficient	R/S ratio
				g ha^{-1}		
S	Diclofop	—	—	969[a]	0.99	
R1		106.8 (4.4)	0.8 (0.3)	> 8,800	0.90	> 9
R2		100.1 (1.9)	0.4 (0.1)	> 8,800	0.98	> 9
R3		103.1 (6.5)	0.7 (0.3)	> 8,800	0.84	> 9
R4		102.5 (3.7)	0.5 (0.2)	> 8,800	0.90	> 9
S	Fenoxaprop-P	109.8 (10.1)	1.1 (0.3)	12 (4.2)	0.92	
R1		101.3 (6.6)	1.1 (0.2)	42 (8.6)	0.97	3.5
R2		103.5 (4.2)	0.9 (0.2)	210 (35)	0.97	17.5
R3		101 (4.7)	1.4 (0.3)	127 (17.8)	0.97	10.6
R4		100.7 (3.2)	0.4 (0.1)	> 308	0.97	> 25
S	Sethoxydim	118.3 (12.7)	1.1 (0.5)	122 (48)	0.90	
R1		113 (9.5)	1.4 (0.4)	375 (90.9)	0.94	3
R2		112.9 (8)	1.3 (0.6)	1,290 (347)	0.90	10.6
R3		118 (10)	1.8 (0.7)	553 (131)	0.91	4.5
R4		108.2 (3.6)	1.7 (0.4)	1,290 (148.7)	0.95	10.6
S	Clethodim	102.7 (8)	0.8 (0.2)	4.7 (1.5)	0.95	
R1		101.5 (12.2)	0.9 (0.3)	10 (4.6)	0.92	2
R2		102.9 (8.3)	1.1 (0.2)	21 (5.2)	0.96	4.5
R3		102.5 (12.6)	0.7 (0.3)	22.5 (11.6)	0.90	4.8
R4		102.2 (6.4)	0.8 (0.2)	39.4 (6.7)	0.96	8.4
S	Pinoxaden	104 (8.8)	1.4 (0.3)	1.4 (0.3)	0.96	
R1		100.5 (5.6)	0.5 (0.1)	7.3 (2.6)	0.97	5
R2		101 (5.5)	0.5 (0.1)	80 (28.6)	0.95	57
R3		100.4 (7.6)	0.4 (0.1)	5.4 (3.4)	0.95	4
R4		102.8 (5.7)	0.8 (0.1)	44 (9.4)	0.97	31.4

[a] The GR_{50} value was calculated from parameters obtained from exponential decay (Equation 2).

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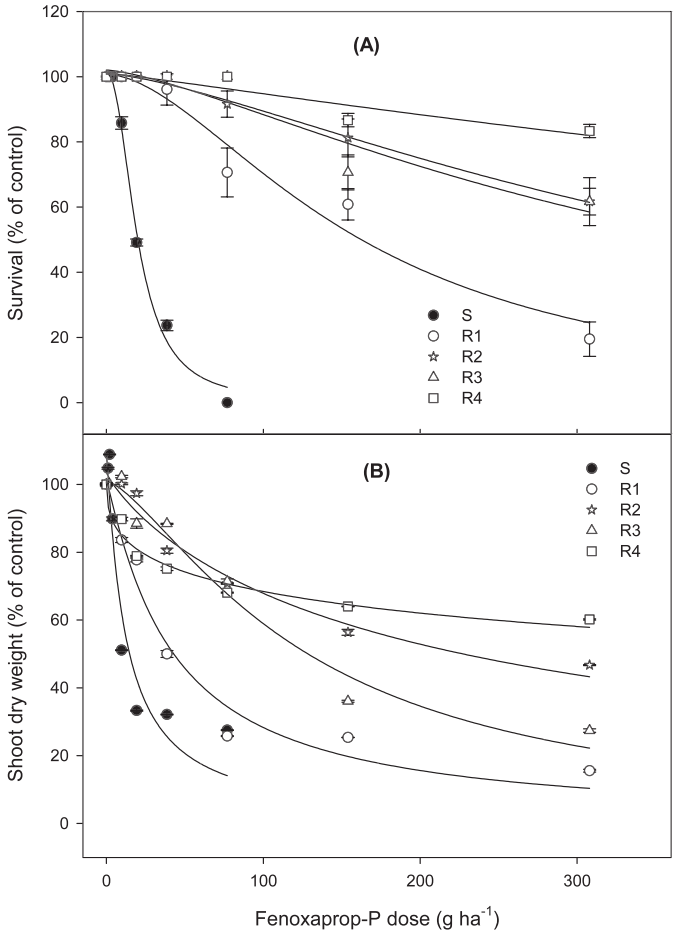

Figure 2. Survival (A) and shoot dry weight (B) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations to fenoxaprop-P 21 d after treatment. Registered or recommended rate for fenoxaprop-P is 38.50 g ha^{-1}. Bars indicate the standard errors of the means of three replicates.

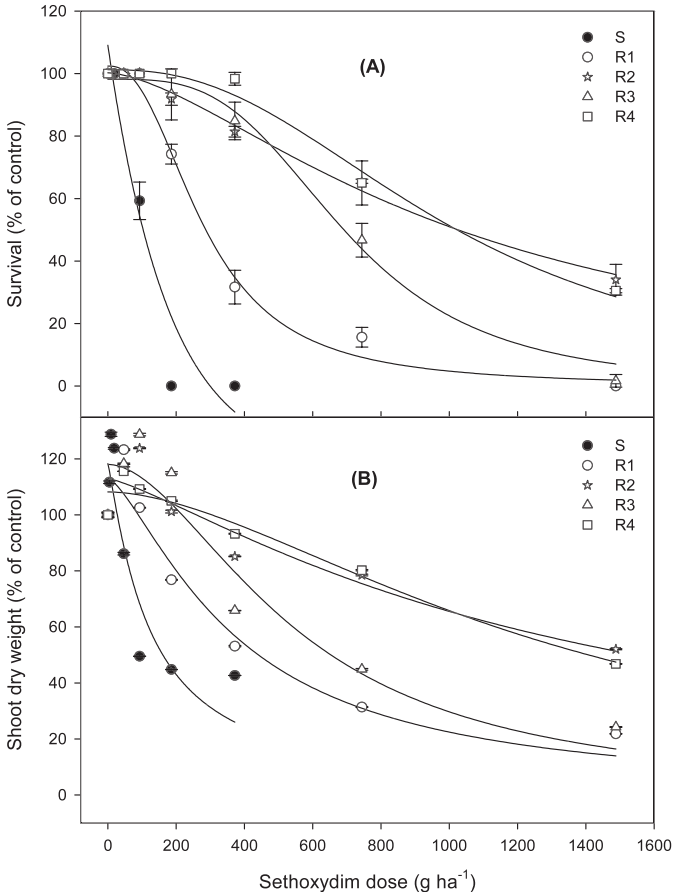

Figure 3. Survival (A) and shoot dry weight (B) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations to sethoxydim 21 d after treatment. Registered or recommended rate for sethoxydim is 186 g ha^{-1}. Bars indicate the standard errors of the means of three replicates.

resistant populations were 3 to 10.5 times higher than the S population (Table 2). Meanwhile, sethoxydim was found to greatly reduce the shoot dry weight of populations R1 and R3 but was less effective on the populations R2 and R4 (Figure 3B). The sethoxydim GR_{50} values for all four resistant populations were 3 to 10.6 times higher than the S population (Table 3).

For the CHD herbicide clethodim, low-level resistance was recorded in populations R1, R2, and R3, whereas R4 exhibited moderate-level resistance (Figure 4A). Resistant populations had clethodim LD_{50} values 2.6- to 6.6-fold higher than the S population (Table 2). Similarly, clethodim was found to reduce the growth of all resistant populations effectively (Figure 4B). The clethodim GR_{50} values for all four resistant populations were 2- to 8.4-fold greater than the S population (Table 3). Similar, lower level clethodim resistance but higher level resistance to other APP and CHD herbicides has been observed in populations of goosegrass (*Eleusine indica* L. Gaertn.), rigid ryegrass (*Lolium rigidum* Gaud.), green foxtail [*Setaria viridis* (L.) Beauv.], giant foxtail (*Setaria faberi* Herrm.), large crabgrass (*Digitaria sanguinalis* L. Scop.), johnsongrass [*Sorghum halepense* (L.) Pers.] biotypes, and *Avena* spp. (Leach et al. 1995; Owen and Powles 2009; Shukla et al. 1997; Stoltenberg and Wiederholt 1995).

Although all four resistant populations had never been selected with the herbicide pinoxaden, low-level resistance in R1 and R3, moderate-level resistance in R2, and high-level resistance in R4 population were recorded to this new ACCase-inhibiting herbicides (Figure 5A). The pinoxaden LD_{50} values of resistant populations were 3.5 to 20 times higher than the S population (Table 2). For shoot dry weight, a large reduction was observed in populations R1 and R3, whereas R2 and R4 were less affected by this herbicide (Figure 5B). The pinoxaden GR_{50} values for the resistant populations were 4 to 57 times higher than the S population (Table 3). Pinoxaden was released in 2006 (approximately 1 yr after these seeds were collected); therefore, resistance to pinoxaden in these populations was selected by the use of other ACCase-inhibiting herbicides. Several, but not all, ACCase resistance mutations are known to confer pinoxaden resistance (Yu et al. 2007).

Overall, the results in this study were similar to the original herbicide screen study, although percent survival at the recommended field rates for some populations was slightly

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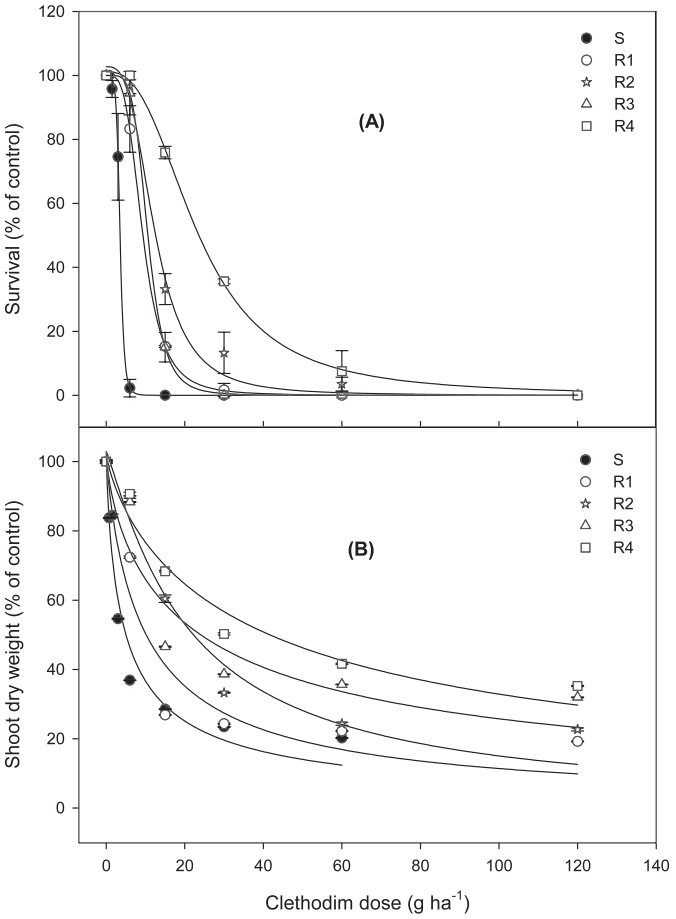

Figure 4. Survival (A) and shoot dry weight (B) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations to clethodim 21 d after treatment. Registered or recommended rate for clethodim is 60 g ha^{-1}. Bars indicate the standard errors of the means of three replicates.

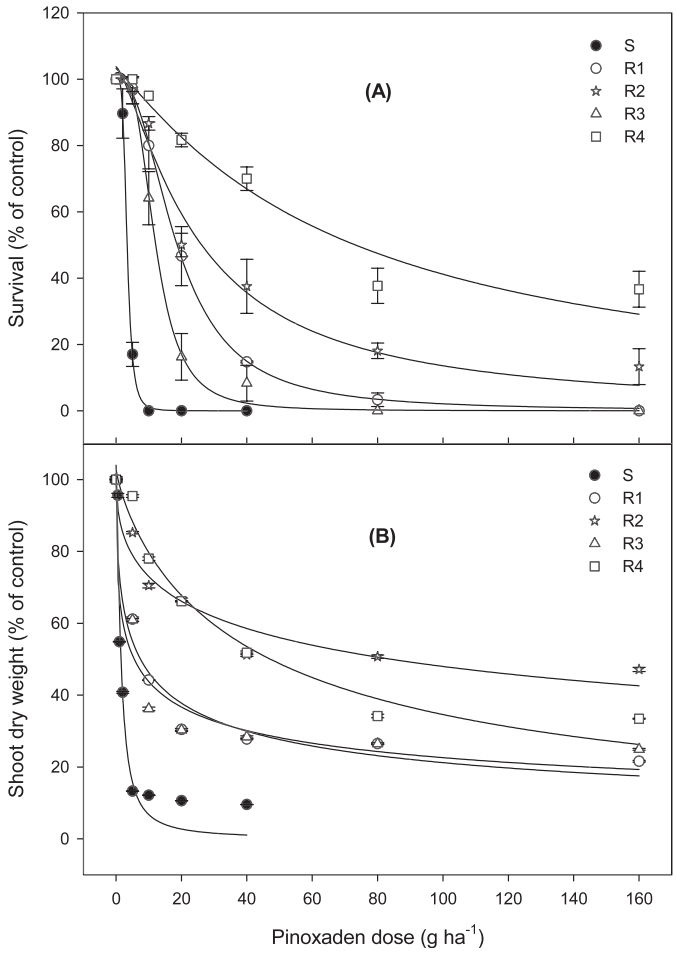

Figure 5. Survival (A) and shoot dry weight (B) of the susceptible (S) and resistant (R1, R2, R3, and R4) *Avena* spp. populations to pinoxaden 21 d after treatment. Registered or recommended rate for pinoxaden is 20 g ha^{-1}. Bars indicate the standard errors of the means of three replicates.

higher (Table 1). The four resistant *Avena* spp. populations in this study exhibited high-level resistance to diclofop, with varying levels of cross-resistance to other APP as well as CHD and PPZ herbicides. Different patterns of resistance have been reported previously in wild oat (Cruz-Hipolito et al. 2011; Heap et al. 1993; Seefeldt et al. 1994) and sterile oat (Mansooji et al. 1992; Uludag et al. 2007) populations. This variation in resistance level and pattern is likely because of the diversity in ACCase mutations and resistance mechanisms. For example, specific resistance-endowing ACCase mutations confer resistance to specific ACCase-inhibiting herbicides (Devine 1997; Yu et al. 2007). Additionally, it is equally possible that non–target-site resistance mechanisms co-exist in these resistant populations. Enhanced rates of diclofop metabolism can confer resistance, and both mechanisms of resistant ACCase and enhanced rates of herbicide metabolism can be present in the same resistant *Avena* spp. individual/population (Maneechote et al. 1997).

This study has revealed resistance across many ACCase-inhibiting herbicides in *Avena* spp. from the WA grain belt. The differences in cross-resistance patterns in these four resistant populations, which are located many kilometers apart, indicate that resistance has evolved independently, and each resistant population has likely undergone different selection pressures (both herbicidal and nonherbicidal). Because the populations have no resistance to other herbicide modes of action (Owen and Powles 2009), it is possible to manage these *Avena* spp. populations by using alternative herbicides (PRE and POST) and herbicide rotation. However, it should be noted that *Avena* spp. populations with resistance to acetolactate synthase–inhibiting herbicides and flamprop have been documented in other Australian cropping regions (Boutsalis 2007; Widderick and Cook 2011). Thus, relying solely on herbicide rotation to control wild oat and delay the evolution of herbicide resistance is not recommended. The use of an integrated weed management approach utilizing a range of herbicide and nonherbicide control practises will minimize the risk for resistance evolution.

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