CSIRO PUBLISHING
www.publish.csiro.au/journals/cp *Crop & Pasture Science*, 2009, **60**, 25–31

Distribution and frequency of herbicide-resistant wild oat (*Avena* spp.) across the Western Australian grain belt

Mechelle J. Owen[A,B] *and Stephen B. Powles*[A]

[A]Western Australian Herbicide Resistance Initiative, School of Plant Biology, University of Western Australia, 35 Stirling Hwy, Crawley, WA 6009, Australia.
[B]Corresponding author. Email: mowen@cyllene.uwa.edu.au

Abstract. In 2005, a random survey was conducted across 14 million hectares of the Western Australian grain belt to establish the frequency and distribution of herbicide-resistant wild oat (*Avena* spp.) in cropping fields. In total, 677 cropping fields were visited, with wild oat populations collected from 150 fields. These wild oat populations were screened with several herbicides commonly used to control this weed. Most of the wild oat populations (71%) were found to contain individuals resistant to the ACCase-inhibiting herbicide diclofop-methyl. Resistance to other ACCase-inhibiting herbicides was markedly lower. Herbicides of alternative modes of action were effective on all wild oat populations. Overall, wild oat resistance to diclofop-methyl was found to be widespread across the Western Australian grain belt, but resistance to other herbicides was relatively low. Therefore, through diversity in herbicide use and with cultural management, it is possible to maintain wild oat populations at a low level and/or minimise herbicide resistance evolution.

Additional keywords: resistance survey, resistance evolution.

Introduction

Wild oats (*Avena* spp.) are among the world's most common and economically damaging weeds of cropping systems. Globally, wild oats are major weeds of cereal crops where they represent a serious economic threat to crop yields because of their competitiveness, staggered germination, crop mimicry (similar phenology), seed shattering, and the ability of seed to persist in the soil seedbank (Medd and Pandey 1990; Jones and Medd 1997). Wild oats often emerge throughout the growing season, yet many individuals mature earlier than the crop species they infest and their seed shatters readily before crop harvest. Early seed dispersal reduces the number of options to remove seed from the cropping system at harvest; therefore, there is almost universal reliance on early post-emergent, crop-selective herbicides to control wild oat in crop fields. Of greatest impact has been diclofop-methyl (since 1978) and subsequent related acetyl-CoA carboxylase (ACCase)-inhibiting herbicides. These ACCase-inhibiting herbicides have been widely used to selectively control wild oat in a range of major crops and their continued and widespread use has resulted in the evolution of herbicide-resistant populations in major crop-growing regions in Australia, North America, and elsewhere in the world (see Owen *et al*. 2007; Heap 2008).

While herbicide resistance is well known in wild oat, there are few studies which have quantified the geographical extent and severity of herbicide resistance across large regions. In Canada, field surveys of wild oat (*Avena fatua*) revealed the extent of resistance to ACCase and acetolactate synthase (ALS)-inhibiting herbicides (Beckie *et al*. 1999, 2004, 2008; Legere *et al*. 2000). Work in eastern Australia reveals wild oat to be common in cropping regions throughout New South Wales and southern Queensland (Whalley and Burfitt 1972), with more recent survey work revealing the development of herbicide-resistant wild oat (Nietschke *et al*. 1996; Broster *et al*. 1998). There is, however, little quantitative information on the extent and frequency of herbicide resistance in Western Australia, although Paterson (1976) found *Avena* spp. to be widely distributed. Previous work has surveyed the frequency and extent of herbicide resistance in *Lolium rigidum* (ryegrass) and *Raphanus raphanistrum* (wild radish) in Western Australia (WA) (Llewellyn and Powles 2001; Owen *et al*. 2007; Walsh *et al*. 2007). It is also important to quantify the extent and severity of the resistance problem in wild oat. Here, we report on a similar wide-scale survey of wild oat across the WA grain belt.

Materials and methods

Seed collection

Several co-existing wild oat species infest field crops. Previous work in the WA grain belt and elsewhere in Australia has revealed that wild oat infestations can comprise mixtures of up to 3 species: *Avena fatua* L., *A. sterilis* L., and *A. barbata* Pott ex Link (Whalley and Burfitt 1972; Paterson 1976). In WA, Paterson (1976) found that *A. barbata* (50%) occurred predominately in the south-west in areas of high rainfall and on roadside locations, while *A. fatua* (47%) occurred predominately in areas of lower rainfall, with a shorter growing season and in cereal crops. *A. sterilis* occurred in a few isolated locations. *A. fatua* and *A. sterilis* subsp. *ludoviciana* (Dur.) G. & M. predominate in the eastern states (Whalley and Burfitt 1972). In the 150 wild oat-infested fields surveyed here, it was evident that there was a mixture of these wild oat species. As it is difficult to visually

 10.1071/CP08178 1836-0947/09/010025

discriminate among the species, hereafter the populations are generically defined as *Avena* spp. (wild oat).

Crop fields were surveyed over an 8-week period (October and November 2005), commencing in the north (where seed maturation occurred first) and progressing southwards just before the 2005 grain harvest. In total, 677 cropping fields were visited across the WA grain belt (Fig. 1), encompassing a 14 Mha region. Crop fields were visited at random, with 5-km intervals (geo-referenced) in each of 15 agronomic zones (Fig. 1) in the grain belt (interspersed pasture fields were not sampled). These agronomic areas are coded as length of growing season zones from north to south (1–5) (based on mean daily temperature), and rainfall regions from west to east (H, high; M, medium; L, low) (based on annual average rainfall). Fields were surveyed by two people walking in an inverted 'V', 100 m into crop fields. Mature wild oat seeds were collected and bulked from many mature plants in the sampling path, and note was taken of the crop species being cultivated in each field. Wild oats were present in 291 fields but were only collected from 150 fields (in 141 fields, fewer than 10 plants were found in the sample path, which was considered insufficient to constitute a representative sample). During sampling, a weed density score was given to each sample field. This was done by visually estimating the number of wild oat plants/m^2 across the sample area (Table 1). Wild oat was generally patchy across a field; however, in some fields (e.g. those with high density), wild oat was uniformly spread across a large area of the crop field. After field collection, the wild oat seed samples were stored in a non-airconditioned glasshouse over summer (December–March) to help relieve seed dormancy (Paterson *et al*. 1976).

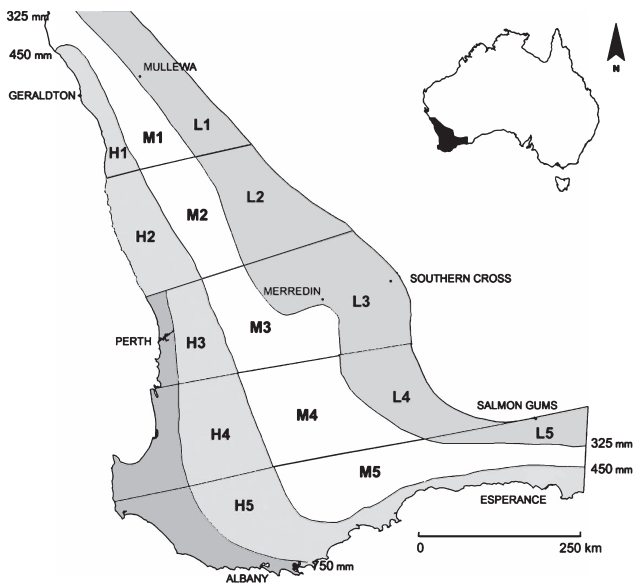

Fig. 1. Map of south-western Western Australia showing the agronomic zones of the grain belt where wild oat samples were collected for herbicide resistance testing. Average annual rainfall isohyets are shown. Rainfall regions are represented by H (high 450–750 mm), M (medium 325–450 mm), and L (low <325 mm). Zones are represented by 1 (north), 2 (north-central), 3 (central), 4 (south-central), and 5 (south).

Resistance testing

During the 2006 and 2007 growing seasons (May–September), seeds of each of the 150 wild oat populations were individually nicked with 2 teeth tweezers to relieve any seed dormancy. Seeds were germinated in 500-mL plastic containers containing agar (1%)-solidified water. One dish containing 100 seeds was germinated for each population and each herbicide. Seed containers were kept at 2−3°C (refrigerator) for a period of 7–10 days until the seeds started to germinate, then the containers were placed at room temperature for 2 days. Fifty seedlings of each population were then transplanted into plastic seedling trays (300 mm by 400 mm by 100 mm) containing potting mix (50% composted pine bark, 25% peat, and 25% river sand). Seedlings were grown outdoors (unless otherwise stated) at the University of Western Australia (UWA) during the normal growing season for this species and were kept well watered and fertilised. For almost all populations, germination (>90%) and seedling survival were high, ensuring that ~45 individual seedlings in each population were screened with each herbicide.

When seedlings reached the 2–3 leaf stage, they were treated with herbicide (upper recommended field rates) (Table 2) using a custom-built, dual-nozzle cabinet sprayer delivering herbicide

Table 1. Number of crop fields surveyed and percentage containing mature wild oat plants at each density classification
Fields were surveyed at the time of full wild oat seed panicle development

Density rating 2005	No. of fields	%
None (no wild oat seed panicles evident)	386	57
Very low (wild oat plants present but difficult to find)	141	21
Low (clearly present but <1 plant/m^2)	83	12
Medium (1–10 plants/m^2)	47	7
High (>10 plants/m^2)	15	2
Very high (crop 'swamped' by wild oat)	5	1
Total	677	100

Table 2. Herbicides and adjuvants applied to wild oat populations during the 2006 and 2007 growing seasons

Chemical class	Active ingredient	Rate (g/ha)	Adjuvants
Aryloxyphenoxy-propionate	Diclofop-methyl	563	0.25% BS 1000
	Fenoxaprop	38.5	0.25% BS 1000
	Clodinafop	18	0.5% Hasten
Cyclohexanedione	Clethodim	60	1% Hasten
	Sethoxydim	186	0.25% BS 1000
	Tralkoxydim	200	0.5% Hasten
Phenylpyrazolin	Pinoxaden	20	0.5% Adigor
Sulfonylurea	Mesosulfuron	9.9	0.25% BS 1000
Imidazolinone	Imazapic + Imazapyr	10.5 + 3.5	0.5% Hasten
Thiocarbamate	Triallate	1000	None
Arylalanine	Flamprop	270	None
Glycine	Glyphosate	810	None

in 100 L/ha water at 210 kPa, at a speed of 3.6 km/h A range of wild oat-active herbicides was used (Table 2) to determine the spectrum of herbicide resistance. Survival was assessed 21 days after treatment. For diclofop-methyl, all surviving seedlings were cut back to a height of 30 mm at 21 days after treatment, allowed to regrow for 5 days, and then treated with the cyclohexanedione ACCase-inhibiting herbicide sethoxydim (Table 2). Sethoxydim was applied to diclofop-methyl survivors because previous work has shown that plants can metabolise diclofop-methyl but not sethoxydim (Tardif and Powles 1994), indicating target site resistance in the plants which survived both herbicide treatments. All ACCase and ALS-inhibiting herbicides were applied at the 2–3 leaf stage and plants were assessed for mortality 21 days after treatment.

For the pre-emergent herbicide triallate, 50 seeds of each population (seeds were pre-germinated and planted when the radicle was just visible) were placed on the soil surface in seedling trays (300 mm by 400 mm by 100 mm) containing potting mix, and treated with triallate (liquid formulation) using the same procedure as described above. Immediately after treatment, 10 mm of untreated soil was placed on the soil surface to prevent triallate volatilisation. Seedling emergence was recorded 21 days after treatment. Only seedlings that had reached the 2–3 leaf stage, and were of comparable growth stage with that of the untreated controls, were recorded as resistant.

For the mid-season treatment (usually applied in early spring), flamprop-methyl was applied to 4-leaf to early tillering wild oat seedlings. After treatment, seedlings were then placed in a controlled-temperature glasshouse (26°C) for a period of 4 weeks because screening was conducted in winter months and the glasshouse reflected the temperatures when this herbicide is normally applied. Assessments were made 28 days after treatment and compared with untreated controls.

Known resistant and susceptible wild oat populations were used as controls for each herbicide treatment. In all experiments, with all herbicides, 100% mortality occurred in the known susceptible population, whereas with the known resistant populations there was always very high survival (>90%) with all herbicides used (data not shown). Herbicide effect was assessed by determining seedling mortality 21 days after herbicide treatment (unless stated otherwise). Wild oat populations were classed as resistant if 20% or more of the individuals in the population survived the herbicide. Where there was 2–19% survival, the population was classed as developing resistance. Where there was less than 2% survival, the population was classed as susceptible. Some populations were not screened with all herbicides due to a small sample size for these populations.

Results and discussion

Areas of wild oat infestation

Despite the almost universal usage of herbicides and other control measures practised annually in crop fields (including the 2005 growing season), wild oat was found to infest many, but by no means all, crop fields across the WA grain belt. The pattern of wild oat infestation varied according to location and rainfall. Of the 677 fields visited, 291 fields (43%) were visually infested with seed-producing wild oat plants. Table 1 shows that only 10% of fields contained seed-producing wild oat plants at a density greater than 1 plant/m^2. Over half (51%) of the wild oat samples came from the H3, M3, L3 regions, with a further 30% of populations coming from the northern areas (Fig. 1). The southern areas, particularly coastal regions, had very low wild oat infestations. Previous work in the WA grain belt (Paterson 1974) also found that the distribution of wild oats was patchy and related to soil type and rainfall. It is emphasised that this survey was conducted very late in the growing season, and almost all fields would have received early-season herbicide treatments to control wild oat infestations (which should have controlled most herbicide-susceptible plants). Therefore the survey reveals only the presence of wild oat plants reaching maturity.

The survey revealed, as expected, that wheat (*Triticum aestivum* L.) was the dominant crop growing in 420 of the 677 fields (62%), with the remainder comprising barley (*Hordeum vulgare* L.) (15%), cultivated oat (*Avena sativa* L.) (8%), lupin (*Lupinus* spp.) (7%), canola (*Brassica napus* L.) (6%), and field pea (*Pisum sativum* L.) (2%). A high proportion (97%) of the wild oat infestations at seed collection were in cereal crops (wheat 71%, barley 20%, cultivated oat 6%), indicating fewer control options available for wild oat in cereal crops compared with canola and lupins.

Resistance to ACCase-inhibiting herbicides

Aryloxyphenoxypropionate herbicides (AOPP)

Screening of the wild oat populations randomly collected from fields in the WA grain belt revealed widespread resistance to the wheat-selective ACCase-inhibiting herbicide diclofop-methyl. Of the 150 wild oat populations sprayed with diclofop-methyl, 16% were found to be resistant, with a further 55% in the developing resistance category (Table 3). In total, 71% of the

Table 3. Percentage of herbicide-resistant wild oat populations in each category for each herbicide

Populations were classed as resistant (20% or more survival), as developing resistance (2–19% survival), or as susceptible (less than 2% survival)

Herbicide	Susceptible	Developing resistance	Resistant	Total resistance	No. of popns screened
Diclofop-methyl	29	55	16	71	150
Fenoxaprop	78	17	5	22	127
Clodinafop	97	0	3	3	129
Sethoxydim[A]	76	12	12	24	103
Tralkoxydim	95	5	0	5	150
Clethodim	97	3	0	3	120
Pinoxaden	97	1.5	1.5	3	147
Mesosulfuron	100	0	0	0	122
Imazapic + imazapyr	100	0	0	0	116
Glyphosate	100	0	0	0	135
Triallate	100	0	0	0	127
Flamprop	100	0	0	0	146

[A]Sethoxydim resistance was only tested on populations with diclofop survivors.

wild oat populations displayed some level of diclofop-methyl resistance, with only 29% of populations remaining susceptible. Most resistant populations were found in the medium- and high-rainfall zones and the central agricultural zones H3 and M3, where wild oats were more prevalent and at higher densities (Table 4, Fig. 1).

The survey revealed that resistance to other ACCase-inhibiting herbicides was markedly lower than that found for diclofop-methyl. Screening of 127 populations with the herbicide fenoxaprop (which is widely used for wild oat control), revealed that only 22% of the populations exhibited some level of resistance (5% classed as resistant, and a further 17% in the developing resistance category). These results contrast with diclofop-methyl, where 71% of populations displayed resistance. This difference is very likely due to the lengthy period of selection pressure imposed from the much greater historical use of diclofop-methyl across the WA grain belt. For example, diclofop-methyl is registered in both cereal and broad-leaf crops (lupin, pea, and canola) for both wild oat and *L. rigidum* control, whereas fenoxaprop is only registered for wild oat control in cereal crops. It is important to recognise that because the grass weed *L. rigidum* (ryegrass) is ubiquitous across this region (Owen *et al*. 2007), diclofop-methyl has been almost universally used to control ryegrass. In contrast, fenoxaprop is only used for wild oat control and there has been much less overall use. This phenomenon is also evident in previous work, where some wild oat populations from Australia have a high level of resistance to diclofop-methyl but only moderate to low resistance to other ACCase-inhibiting herbicides (Mansooji *et al*. 1992; Maneechote *et al*. 1997). Several different mutations of ACCase (Yu *et al*. 2007) and non-target site-enhanced metabolism (Maneechote *et al*. 1997) are known to endow diclofop-methyl resistance.

For the ACCase-inhibiting herbicide clodinafop, nearly all the wild oat populations were susceptible. Of the 129 populations screened with clodinafop, 125 populations (97%) were susceptible, with only 4 populations (3%) classed as resistant. It is noteworthy that these few resistant populations (WAA1, WAA2, WAA3, WAA4) were also resistant to many other ACCase-inhibiting herbicides tested (Table 5).

Among individual field populations, there were large differences in the number of individuals surviving each herbicide, with resistance to diclofop-methyl the highest and clodinafop showing a similar level of control to the cyclohexanedione class of ACCase-inhibiting herbicides (see below). These differences are almost certainly due to different resistance mechanisms (enhanced metabolism and/or one or more different ACCase mutations), which will be present in different *Avena* populations (Maneechote *et al*. 1994; Wenjie *et al*. 2004). For example, the presence of different ACCase mutations can each confer a different pattern and level of resistance to various ACCase herbicides within or between populations (Devine 1997; Yu *et al*. 2007). Although the mechanism(s) endowing diclofop-methyl resistance cannot be determined from these whole-plant studies, we expect diversity in resistance mechanisms/mutations. Previously it has been identified that mutations of the ACCase gene or non-target site-enhanced metabolism can confer resistance to diclofop-methyl, and that both mechanisms can be present in the same resistant *Avena* individual (Maneechote *et al*. 1997).

Cyclohexanedione herbicides (CHD)

All wild oat plants that survived diclofop-methyl were treated with the ACCase-inhibiting CHD herbicide sethoxydim. Plants are generally not able to metabolise sethoxydim, therefore resistance to this herbicide is likely due to target-site ACCase gene mutation(s) (Tardif and Powles 1994). Of the 103 populations assessed as diclofop-methyl-resistant or developing resistance, only 12 (12%) of these populations were classified as resistant to sethoxydim, with another 12 populations in the developing resistance category (Table 3).

For the CHD herbicide tralkoxydim, 95% of the tested wild oat populations were found to be susceptible. Only 8 (5%)

Table 4. Percentage of wild oat populations that are: resistant (R), developing resistance (DR), and susceptible (S) to AOPP and CHD herbicides from each agronomic zone

Refer to Fig. 1 for agronomic zones

Zone	Diclofop			Fenoxaprop			Clodinafop			Sethoxydim			Tralkoxydim			Clethodim			Pinoxaden		
	R	DR	S	R	DR	S	R	DR	S	R	DR	S	R	DR	S	R	DR	S	R	DR	S
H1	0	33	67	0	14	86	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
M1	0	12	88	0	17	83	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
L1	0	17	83	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
H2	50	50	0	0	0	100	0	0	100	0	50	50	0	0	100	0	0	100	0	0	100
M2	22	45	33	0	50	50	0	0	100	0	0	100	0	22	78	0	0	100	0	0	100
L2	9	64	27	9	27	64	0	0	100	13	13	74	0	0	100	0	0	100	0	0	100
H3	13	54	33	0	23	77	0	0	100	10	10	80	0	7	93	0	0	100	0	0	100
M3	36	46	18	20	20	60	20	0	80	36	14	50	0	9	91	0	17	83	5	9	86
L3	18	64	18	6	31	63	0	0	100	0	21	79	0	12	88	0	0	100	0	0	100
H4	17	83	0	0	0	100	0	0	100	0	25	75	0	0	100	0	0	100	0	0	100
M4	6	63	31	0	7	93	0	0	100	9	0	91	0	0	100	0	0	100	0	0	100
L4	25	75	0	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
H5	0	100	0	0	0	100	0	0	100	50	0	50	0	0	100	0	0	100	0	0	100
M5	23	69	8	8	8	84	8	0	92	25	8	67	0	8	92	0	9	91	8	0	92
L5	0	75	25	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100

Table 5. Distribution and frequency of herbicide resistance response among selected populations of wild oat collected from the Western Australian grain belt in 2005

These populations were selected to show the variation in cross-resistance patterns among wild oat populations collected from different crop fields

Herbicide	WAA1	WAA2	WAA3	WAA4	WAA5	WAA6	WAA7	WAA8	WAA9	WAA10	WAA11
Diclofop	R	R	R	R	R	R	R	R	DR	DR	R
Fenoxaprop	R	R	R	R	S	S	DR	R	DR	S	R
Clodinafop	R	R	R	R	S	S	S	S	S	S	S
Sethoxydim	R	R	R	R	R	S	DR	S	S	S	S
Tralkoxydim	S	DR	S	DR	S	S	S	DR	DR	DR	S
Clethodim	S	DR	R	DR	S	S	S	S	S	S	S
Pinoxaden	DR	R	DR	R	S	S	S	S	S	S	S
Mesosulfuron	S	S	S	S	S	S	S	S	S	S	S
Imazapic + Imazapyr	S	S	S	S	S	S	S	S	S	S	S
Glyphosate	S	S	S	S	S	S	S	S	S	S	S
Triallate	S	S	S	S	S	S	S	S	S	S	S
Flamprop	S	S	S	S	S	S	S	S	S	S	S

populations were classed as developing resistance to tralkoxydim. These CHD-resistant populations tended to come from the north-central and central grain belt (H2, H3, M2, M3, L2, L3) and the higher rainfall zones in the south (H4 and M5) (Fig. 1, Table 4). Wild oat populations were also treated with the ACCase-inhibiting CHD herbicide clethodim. Of the 120 populations treated with clethodim, only 3 populations (3%) were classed as resistant or developing resistance, with all other remaining populations being susceptible. These 3 populations (WAA2, WAA3, WAA4) were resistant to most of the other ACCase herbicides tested (Table 5).

Overall, CHD ACCase-inhibiting herbicide resistance was low, with the clethodim-resistant populations confined to the M3 and M5 zones. The central zones were where wild oat populations were most common and at high densities (Fig. 1). There were 4 populations (WAA1, WAA2, WAA3, WAA4) that were resistant to most ACCase-inhibiting herbicides tested (Table 5). As with the AOPP herbicides, there were marked differences in the level of resistance among CHD herbicides. Similar diversity in levels of herbicide resistance was evident among wild oat populations collected from the Red River Valley of Minnesota and North Dakota, USA (Mengistu *et al*. 2003), and from cereal-growing regions of Turkey (Uludag *et al*. 2007). It must be recognised that crop fields receive different herbicide (and other) wild oat control treatments and therefore each field receives a somewhat unique evolutionary selection pressure. Thus, especially as wild oat is self-pollinated (gene flow by pollen is very limited), individual field wild oat populations under herbicide selection will differ from each other (unless there is seed movement).

Explanations for the differences in levels of resistance frequency to different ACCase-inhibiting herbicides must be made with caution. Previous work with ACCase herbicide resistance in *Lolium rigidum* establishes that the level of phenotypic resistance is dependent upon the rate and type of ACCase herbicide used, as well as the specific resistance mechanism and whether individuals are heterozygous or homozygous for the resistance mechanism (Yu *et al*. 2007). Only by studying each population at the genetic and molecular level can these differences be elucidated.

Pinoxaden (Dens)

The survey revealed that resistance is rare to the recently commercialised (2006) ACCase-inhibiting herbicide pinoxaden. Only 4 (3%) populations had pinoxaden resistance, with 2 populations classed as resistant and a further 2 populations developing resistance. These populations came from the M3 and M5 zone (Fig. 1, Table 4) and were resistant to most other ACCase-inhibiting herbicides tested (Table 5). Overall, 97% of the populations were found to be susceptible to pinoxaden. The resistance results to the den herbicide pinoxaden were very similar to those for clethodim and clodinafop. Again, the same 4 populations were resistant to 3 chemically dissimilar ACCase-inhibiting herbicides (Table 5). As pinoxaden had never been used in Australia at the time of this survey, pinoxaden resistance had been selected by the use of other ACCase-inhibiting herbicides. Several but not all ACCase mutations are known to confer pinoxaden resistance (Yu *et al*. 2007). Clearly, selection with earlier ACCase herbicides has resulted in the enrichment of ACCase mutation(s) that confer cross-resistance to pinoxaden.

Cross-resistance patterns across ACCase-inhibiting herbicides

The frequency of herbicide resistance across different ACCase-inhibiting herbicides was found to be variable. Many populations (43%) were found to be resistant only to the ACCase-inhibiting herbicide diclofop-methyl (i.e. they showed no resistance to the other herbicides tested). However, nearly one-third (28%) of the diclofop-methyl-resistant populations displayed resistance to other ACCase-inhibiting herbicides, whereas any wild oat populations with resistance to other ACCase-inhibiting herbicides were always resistant to diclofop-methyl. If a population was susceptible to diclofop-methyl, it was susceptible to all other ACCase herbicides tested. Cross-resistance, while being variable across the ACCase-inhibiting herbicides, was greater for the AOPP than for the CHD herbicides. Approximately 16% of wild oat populations were cross-resistant to the ACCase-inhibiting herbicides diclofop-methyl and fenoxaprop (and diclofop-methyl and sethoxydim), whereas

only 6% of wild oat populations were resistant to both fenoxaprop and sethoxydim. Less than 3% of wild oat populations were cross-resistant to 2 or more CHD herbicides (data not shown).

No resistance to ALS-inhibiting herbicides

The wild oat populations were screened with two ALS-inhibiting herbicides, mesosulfuron from the sulfonylurea class and imazapic + imazapyr from the imidazolinone class. There were 116 populations treated with the herbicide imazapic + imazapyr, and 122 populations were treated with the sulfonylurea herbicide mesosulfuron. All wild oat populations were found to be susceptible to these ALS-inhibiting herbicides (Table 3).

No resistance to herbicides with three other modes of action

Populations were treated with the herbicide glyphosate, the pre-emergent herbicide triallate, and the mid-season herbicide flamprop-methyl (Table 3). All populations treated with these herbicides were susceptible at field rates, indicating that wild oat populations with ACCase resistance are effectively controlled by these different herbicide modes of action.

Comparison with Lolium rigidum *(ryegrass) from the same region*

The frequency of resistance in wild oat found in this random survey across the WA grain belt contrasts with the much greater frequency and extent of resistance found in ryegrass across the same region. Wild oat was found to be present in far fewer fields, and resistance frequency was lower than in ryegrass, which was found to be present in almost all fields and almost always resistant (Llewellyn and Powles 2001; Owen *et al*. 2007). Much greater resistance in ryegrass is understandable because ryegrass is a highly genetically variable, cross-pollinated species present at very high numbers across vast areas (it has been widely seeded as pasture throughout Australian grain-growing regions) (Powles and Matthews 1992). These factors favour ryegrass resistance evolution in comparison with wild oat, which is present in much lower numbers across limited areas, has less genetic variability, and is self-pollinated (which may help to decrease the spread of resistance due to reduced gene flow via pollen). Self-pollinated species often have less within-population variability due to lack of gene flow among plants (Murray *et al*. 2002), but can also have a much greater among-population variability as they are usually highly variable genetically (Allard 1996). This is due to genetic isolation, especially when individual populations are under different selection pressures due to management strategies. These biological and numerical differences between wild oat and ryegrass help explain why resistance is greater in ryegrass (Powles and Matthews 1992) than in self-pollinated wild oats.

Future implications

This survey has revealed widespread wild oat resistance to the ACCase-inhibiting herbicide diclofop-methyl across the vast WA grain belt. However, resistance to other AOPP and CDH ACCase herbicides was relatively low. Alternative ACCase-inhibiting herbicides clodinafop, clethodim, and pinoxaden remain effective on 97% of the wild oat populations tested. Importantly, herbicides of other modes of action such as ALS-inhibiting herbicides, glyphosate, triallate, and flamprop, also remain effective in controlling the ACCase herbicide-resistant wild oat populations. Thus, currently there is the opportunity to control wild oat by selecting from a diverse range of herbicide modes of action, acting at different stages of crop growth (pre-seeding, seeding, post-seeding, and late stem elongation). However, there should not be complacency, as previous work has identified *Avena* populations that have developed resistance to ALS-inhibiting herbicides (Beckie *et al*. 1999; Mengistu *et al*. 2003), triallate (Kern *et al*. 1996), and flamprop-methyl (Friesen *et al*. 2000; Broster 2004). Nevertheless, the availability of herbicide diversity means that herbicide resistance evolution in wild oat can be minimised through diverse herbicide usage and rotation, together with cultural management practices. The incidence of wild oat in crop fields can be managed by controlling *Avena* patches, as the patches do not spread rapidly (Thill and Mallory-Smith 1997; Murray *et al*. 2002). By minimising seed set during the spring (e.g. cutting the crop early and baling for hay, which prevents wild oat from setting viable seed), the number of potentially resistant seeds being allowed back into the seedbank can be reduced. It is important to manage wild oat populations through diverse cropping/farming programs, diverse herbicide use, and integrated management strategies to sustain the efficacy of these herbicides and farming systems in the long-term.

Acknowledgments

We thank WAHRI staff and in particular Roslyn Owen, Aaron Gates, Emma Glasfurd, and Fiona Van Rijnswoud, who provided invaluable technical assistance in many areas of the research that contributed to this paper. We are grateful to the GRDC for providing funding for this research.

References

Allard RW (1996) Genetic basis of the evolution of adaptedness in plants. *Euphytica* **92**, 1–11. doi: 10.1007/BF00022822

Beckie HJ, Hall LM, Meers S, Laslo JJ, Stevenson FC (2004) Management practices influencing herbicide resistance in wild oat. *Weed Technology* **18**, 853–859. doi: 10.1614/WT-03-124R

Beckie HJ, Leeson JY, Thomas AG, Brenzil CA, Hall LM, Holzgang G, Lozinski C, Shirriff S (2008) Weed resistance monitoring in the Canadian prairies. *Weed Technology* **22**, 530–543. doi: 10.1614/WT-07-175.1

Beckie HJ, Thomas AG, Legere A, Kelner DJ, Van Acker RC, Meers S (1999) Nature, occurrence, and cost of herbicide-resistant wild oat (*Avena fatua*) in small-grain production areas. *Weed Technology* **13**, 612–625.

Broster JC (2004) A population of wild oats (*Avena ludoviciana* Durieu) resistant to flamprop-m-methyl. In 'Proceedings of the 14th Australian Weeds Conference: Weed Management—Balancing People, Planet, Profit'. Wagga Wagga, NSW. (Eds BM Sindel, SB Johnson) pp. 432–433. (Weed Society of NSW Inc.)

Broster JC, Pratley JE, Slater PD, Medd RW (1998) Herbicide resistance in wild oats in southern New South Wales. In 'Proceedings of the 9th Australian Agronomy Conference'. pp. 579–582. (The Australian Society of Agronomy: Wagga Wagga, NSW)

Devine MD (1997) Mechanisms of resistance to acetyl-Coenzyme A carboxylase inhibitors: a review. *Pesticide Science* **51**, 259–264. doi: 10.1002/(SICI)1096-9063(199711)51:3<259::AID-PS644>3.0.CO;2-S

Friesen LF, Jones TL, Van Acker RC, Morrison IN (2000) Identification of *Avena fatua* populations resistant to imazamethabenz, flamprop, and fenoxaprop-P. *Weed Science* **48**, 532–540. doi: 10.1614/0043-1745(2000)048[0532:IOAFPR]2.0.CO;2

Heap IM (2008) International survey of herbicide resistant weeds. Herbicide Resistance Action Committee and Weed Science Society of America. Available at: www.weedscience.com

Jones R, Medd R (1997) Economic analysis of integrated management of wild oats involving fallow, herbicide and crop rotational options. *Australian Journal of Experimental Agriculture* **37**, 683–691. doi: 10.1071/EA97036

Kern AJ, Peterson DM, Miller EK, Colliver CC, Dyer WE (1996) Triallate resistance in *Avena fatua* L. is due to reduced herbicide activation. *Pesticide Biochemistry and Physiology* **56**, 163–173. doi: 10.1006/pest.1996.0070

Legere A, Beckie HJ, Stevenson FC, Thomas AG (2000) Survey of management practices affecting the occurrence of wild oat (*Avena fatua*) resistance to acetyl-CoA carboxylase inhibitors. *Weed Technology* **14**, 366–376. doi: 10.1614/0890-037X(2000)014[0366:SOMPAT]2.0.CO;2

Llewellyn RS, Powles SB (2001) High levels of herbicide resistance in rigid ryegrass (*Lolium rigidum*) in the wheat belt of Western Australia. *Weed Technology* **15**, 242–248. doi: 10.1614/0890-037X(2001)015[0242:HLOHRI]2.0.CO;2

Maneechote C, Holtum JAM, Preston C, Powles SB (1994) Resistant Acetyl-CoA Carboxylase is a mechanism of herbicide resistance in a biotype of *Avena sterilis* ssp. *ludoviciana. Plant & Cell Physiology* **35**, 627–635.

Maneechote C, Preston C, Powles S (1997) A diclofop-methyl resistant *Avena sterilis* biotype with a herbicide resistant acetyl-coenzyme A carboxylase and enhanced metabolism of diclofop-methyl. *Pesticide Science* **49**, 105–114. doi: 10.1002/(SICI)1096-9063(199702)49:2<105::AID-PS507>3.0.CO;2-3

Mansooji AM, Holtum JAM, Boutsalis P, Matthews JM, Powles SB (1992) Resistance to aryloxyphenoxypropionate herbicides in two wild oat species (*Avena fatua* and *Avena sterilis* ssp. *ludoviciana*). *Weed Science* **40**, 599–605.

Medd RW, Pandey S (1990) Estimating the cost of wild oats (*Avena* spp.) in the Australian wheat industry. *Plant Protection Quarterly* **5**, 142–144.

Mengistu LM, Messersmith CG, Christoffers MJ (2003) Diversity of herbicide resistance among wild oat sampled 36 yr apart. *Weed Science* **51**, 764–773. doi: 10.1614/0043-1745(2003)051[0764:DOHRAW]2.0.CO;2

Murray BG, Morrison AN, Friesen LF (2002) Pollen-mediated gene flow in wild oat. *Weed Science* **50**, 321–325. doi: 10.1614/0043-1745(2002)050[0321:PMGFIW]2.0.CO;2

Nietschke BS, Llewellyn RS, Reeves TG, Matthews JM, Powles SB (1996) Herbicide resistance in wild oats and annual ryegrass. In 'Proceedings of the 8th Australian Agronomy Conference'. (Ed. M Ashgar) p. 691. (The Australian Society of Agronomy: Toowoomba, Qld)

Owen MJ, Walsh MJ, Llewellyn R, Powles SB (2007) Widespread occurrence of multiple herbicide resistance in Western Australian annual ryegrass (*Lolium rigidum*) populations. *Australian Journal of Agricultural Research* **58**, 711–718. doi: 10.1071/AR06283

Paterson JG (1974) The distribution and ecology of wild oats (*Avena* spp.) in the agricultural environment of Western Australia. PhD Thesis, The University of Western Australia, Australia.

Paterson JG (1976) The distribution of *Avena* species naturalized in Western Australia. *Journal of Applied Biology* **13**, 257–264.

Paterson JG, Goodchild NA, Boyd WJR (1976) Effect of storage temperature, storage duration and germination temperature on the dormancy of seed of *Avena fatua* L. and *Avena barbata.* Pott ex Link. *Australian Journal of Agricultural Research* **27**, 373–379. doi: 10.1071/AR9760373

Powles SB, Matthews JM (1992) Multiple herbicide resistance in annual ryegrass (*Lolium rigidum*): a driving force for the adoption of integrated weed management. In 'Resistance '91: achievements and developments in combating pesticide resistance'. (Eds I Denholm, AL Devonshire, DW Hollomon) pp. 75–87. (Elsevier Publishing: Amsterdam)

Tardif FJ, Powles SB (1994) Herbicide multiple-resistance in a *Lolium rigidum* biotype is endowed by multiple mechanisms: isolation of a subset with resistant acetyl-CoA carboxylase. *Physiologia Plantarum* **91**, 488–494. doi: 10.1111/j.1399-3054.1994.tb02978.x

Thill DC, Mallory-Smith CA (1997) The nature and consequence of weed spread in cropping systems. *Weed Science* **45**, 337–342.

Uludag A, Nemli Y, Tal A, Rubin B (2007) Fenoxaprop resistance in sterile wild oat (*Avena sterilis*) in wheat fields in Turkey. *Crop Protection* **26**, 930–935. doi: 10.1016/j.cropro.2006.08.012

Walsh MJ, Owen MJ, Powles SB (2007) Frequency and distribution of herbicide resistance in *Raphanus raphanistrum* populations randomly collected across the Western Australia wheatbelt. *Weed Research* **47**, 542–550. doi: 10.1111/j.1365-3180.2007.00593.x

Wenjie L, Harrison D, O'Donnell C, Adkins S, Williams R (2004) Molecular characterisation of resistance to ACCase-inhibiting herbicides in wild oat in the northern grain-growing region of Australia. In 'Proceedings of the 14th Australian Weeds Conference: Weed Management—Balancing People, Planet, Profit'. Wagga Wagga, NSW. (Eds BM Sindel, SB Johnson) pp. 421–424. (Weed Society of NSW Inc.)

Whalley RDB, Burfitt JM (1972) Ecotypic variation in *Avena fatua* L., *A. sterilis* L. (*A. ludoviviana*), and *A. barbarta* Pott. in New South Wales and Southern Queensland. *Australian Journal of Agricultural Research* **23**, 799–810. doi: 10.1071/AR9720799

Yu Q, Collavo A, Zheng M, Owen MJ, Sattin M, Powles SB (2007) Diversity of acetyl-coenzyme A carboxylase mutations in resistant *Lolium* populations: evaluation using clethodim. *Plant Physiology* **145**, 547–558. doi: 10.1104/pp.107.105262

Manuscript received 27 May 2008, accepted 24 October 2008