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The frequency of herbicide-resistant wild oat (*Avena* spp.) populations remains stable in Western Australian cropping fields

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Abstract. Avena is a problematic weed of cropping regions of southern Australia and many areas of the world. In 2010, a random survey was conducted across 14 million hectares of the Western Australian grain belt to monitor the change in herbicide resistance levels by comparing resistance frequency results with a survey conducted in 2005. Screening Avena populations with herbicides commonly used to control this weed revealed that 48% of Avena populations displayed resistance to the commonly used acetyl-Co A carboxylase-inhibiting herbicides, which was lower than that found in 2005 (71%). The broad-spectrum herbicides glyphosate and paraquat provided good control of all Avena populations. Resistance to acetolactate synthase-inhibiting herbicides and to flamprop were detected for the first time in Western Australia in this survey. Therefore, a wide range of weed management options that target all phases of the cropping program are needed to sustain these cropping systems in the future.

Additional keywords: resistance survey, resistance evolution, weeds.

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Introduction

Avena (wild oat) is a self-pollinated temperate crop weed that has major adverse economic impacts on crop production, worldwide. Avena is highly competitive, mimics the crops in which it grows, exhibits seed dormancy (staggered germination) and produces many seeds, allowing it to persist in cropping fields (Jones and Medd 1997). In Australia, the incidence of Avena is widespread across the cropping regions, especially in cropping areas of north-eastern Australia (Osten et al. 2007). In southern New South Wales Avena spp. was found in 72% of crop fields in 1993 (Lemerle et al. 1996), and 63% in 2010 (Broster et al. 2012b). In Western Australia, Avena spp. was found in 54% of cropping fields in 1998, 43% in 2005 and 36% of fields in 2008 (Owen and Powles 2009; Borger et al. 2012). Earlier work found Avena spp. to be more widely distributed in Western Australia (Paterson 1976).

Herbicides are the dominant method of weed control in most grain crops. However, herbicide over-reliance has led to the evolution of herbicide-resistant weed populations. Herbicide-resistant biotypes from 244 weed species have been documented from 66 countries, with weeds evolving resistance to 22 of the 25 known modes of action (Heap 2015).

The acetyl-Co A carboxylase (ACCase)- and acetolactate synthase (ALS)-inhibiting herbicides have been widely used to selectively control *Avena* in a range of major crops, and their continued and widespread use has resulted in the evolution of herbicide-resistant populations across several continents (Heap 2015). In Canada, field surveys of *A. fatua* revealed widespread

resistance to ACCase- and ALS-inhibiting herbicides (Beckie et al. 1999, 2004, 2008; Légère et al. 2000). In Australia, resistance to the ACCase-inhibiting herbicides (Mansooji et al. 1992; Owen and Powles 2009; Broster et al. 2011; Ahmad-Hamdani et al. 2012), the ALS-inhibiting herbicides, and flamprop (Heap 2015) has been reported in Avena spp.

In 2005, a large-scale random survey was conducted to determine the distribution and frequency of herbicide resistance in *Avena* populations across the Western Australian grain belt. The survey established widespread resistance to the ACCase-inhibiting herbicide diclofop-methyl; however, no resistance was detected to other herbicide modes of action (Owen and Powles 2009). Here we report on a second (2010) large-scale random survey of *Avena* spp. across the same region of the Western Australian grain belt to update and quantify the geographic extent and spectrum of herbicide resistance.

Materials and methods

Seed collection

Avena seeds were collected as part of a broad-scale survey evaluating herbicide resistance in crop fields for key weed species (Owen et al. 2014). Briefly, farmers were contacted at random through a range of media options (telephone, email, field days) to seek permission and provision of farm maps for surveying. Seed collection was conducted just before crop harvest with crop fields chosen randomly on farms and sampled by two people walking in an inverted 'W' pattern across fields. Avena seeds were collected from a large number

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of mature plants (20-50 plants per field) and bulked at the time of collection. In fields where less than 10 plants were sighted, no sample was collected. Avena was generally present as patchy infestations across crop fields; however, in some cases (e.g. those with high weed density) Avena was uniformly spread across a large area of a crop field. During sampling, weed density was recorded by visually estimating the number of Avena plants across the sample area and recording into category-based classifications (see Results section). Despite dry conditions at the start of the 2010 season, most fields were treated with herbicides; however, some fields were not treated due to economic constraints (David Thomas, pers. comm.). After collection, Avena seed was cleaned and chaff material was separated by aspiration. Seed samples were stored in a warm, dry glasshouse from December to April to after-ripen (Paterson et al. 1976).

Seed germination

During the months of May-October 2012, seeds from each Avena population 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. Seed containers were kept at 4°C (refrigerator) for a period of 10-12 days until the seeds started to germinate, then were placed at room temperature for 3 days. Fifty seedlings from each population were 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) for each herbicide. Known herbicide-susceptible and -resistant populations were used as controls for all herbicide treatments (Owen and Powles 2009). Seedlings were grown outdoors (unless otherwise stated) at the University of Western Australia during the normal growing season for this species and were watered and fertilised as required. During the growing season of 2014, some populations were re-treated with flamprop to confirm their resistance status.

Herbicide resistance screening

To determine the herbicide resistance spectrum, seedlings were treated at the 2-3-leaf stage with a range of herbicides using the upper recommended field rates (Table 1) for Avena in Australia. Herbicides were applied together with appropriate adjuvants

using a custom built dual nozzle (TeeJet XR11001 flat fan, Springfield, IL, USA) cabinet sprayer. Herbicide resistance status was tested for diclofop-methyl (Hoegrass, 500 g a.i. L⁻¹, Bayer CropScience, Australia), fenoxaprop (Wildcat, 110 g a.i. L⁻¹, Bayer CropScience, Hawthorn, Vic., Australia), pinoxaden (Axial, 100 g a.i. L⁻¹, Syngenta, North Ryde, NSW, Australia), sethoxydim (Sertin, 186 g a.i. L⁻¹, Bayer CropScience, Australia), tralkoxydim (Achieve, 400 g a.i. KG⁻¹, Syngenta, Australia), mesosulfuron (Atlantis, $30 \,\mathrm{g}$ a.i. L^{-1} , Bayer CropScience, Australia), imazamox + imazapyr (Intervix, 33 + 15 g a.i. KG⁻¹, Nufarm, Melbourne, Australia), triallate (Avadex Xtra 500 g a.i. L⁻¹, Nufarm, Australia), paraquat (Gramoxone 250 g a.i. L⁻¹, Syngenta, Australia), flamprop (Mataven 90 g a.i. L⁻¹, Nufarm, Australia) and glyphosate (Roundup Powermax 540 g a.i L⁻¹, Nufarm, Australia) (Table 1).

All seedlings surviving diclofop-methyl were cut back to a height of 30 mm at 21 days after treatment, allowed to re-grow for 5 days, and were then treated with sethoxydim (Owen and Powles 2009). This was done to allow comparison with our previous survey (Owen and Powles 2009) and because previous work has shown that plants can metabolise diclofopmethyl but not sethoxydim (Tardif and Powles 1994), indicating possible target site resistance in the plants which survive both herbicide treatments.

For the pre-emergent herbicide triallate, 50 seeds (pregerminated on agar and planted when the radicle was just visible) of each population were placed on the soil surface in seedling trays containing potting mix, and treated with triallate using the same spraying 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.

The mid-season herbicide flamprop-methyl (usually applied in early spring) was applied to 4-leaf to early tillering Avena seedlings. After treatment, seedlings were then placed in a controlled-temperature glasshouse (26°C) for a period of 4 weeks, corresponding to field temperatures at the time when this herbicide is normally applied. Assessments were made 28 days after treatment and treated plants were compared with

Table 1. Herbicides and rates used for resistance screening of Avena populations collected in 2010 from the Western Australian grain belt

Chemical class	Mode of action	Active ingredient	Field rate (g ha ⁻¹)
Aryloxyphenoxy-propionate	Inhibitors of acetyl-Co A carboxylase	Diclofop	500
	Inhibitors of acetyl-Co A carboxylase	Fenoxaprop	38.5
Phenylpyrazoline	Inhibitors of acetyl-Co A carboxylase	Pinoxaden	20
Cyclohexanedione	Inhibitors of acetyl-Co A carboxylase	Tralkoxydim	200
•	Inhibitors of acetyl-Co A carboxylase	Sethoxydim	186
Sulfonylurea	Inhibitors of acetolactate synthase	Mesosulfuron	9.9
Imidazolinone	Inhibitors of acetolactate synthase	Imazamox + Imazapyr	13 + 6
Glycine	Inhibitors of 5-enolpyruvylshikimate -3-phosphatesynthase	Glyphosate	705
Bipyridyl	Inhibitors of photosystem I	Paraquat	250
Arylalanine	Inhibitors of mitosis	Flamprop-methyl	270
Thiocarbamate	Inhibitors of lipid synthesis	Triallate	1000

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untreated controls and known resistant and susceptible populations; putative survivors were allowed to continue through to reproductive stage.

Plant mortality was assessed 21 days after treatment (except where stated), by determining if the growing point was chlorotic or new growth was visible. Plants treated with flamprop were assessed on whether they progressed to the reproductive stage. For some populations only a small number of individuals (five plants or less) survived the herbicide treatment, so these plants were cut back and re-sprayed to further confirm their resistance status. Selected populations were also used as untreated controls, with no mortality observed in these over the duration of the experiments. Known susceptible and resistant Avena biotypes (Owen and Powles 2009) were used as controls in all experiments, with 100% control achieved with the known susceptible population and high survival (>90%) of the known resistant populations occurring under all herbicide treatments (data not shown). In cases where the seed quantity for a particular population was low, some herbicide treatments were omitted.

Data analyses

Each sample collected has been considered here as a separate population, as each field has its own unique micro-environment based on climate, cropping history and soil conditions. Populations were classified based on their percentage survival. Susceptible populations were classified as those having 0% plant survival whereas populations with resistant survivors were classified into two groups: those with >20% plant survival, and those having between 1% and 19% survival. This enables comparisons with our previous survey (Owen and Powles 2009), which employed the two categories of resistance described above, and thus allows us to document the change in resistance over a 5-year period. Farmers often visually recognise resistance at a level of ~20% survival in the field (although this depends on many factors including plant density), at which point they may stop using the herbicide or consider alternative management options. Populations classified with <20% plant survival are unlikely to see commercial failure (>20% survival) until population numbers or resistance increase. Although the present study does not allow direct comparisons between 2005 and 2010 within the same fields, it allows monitoring of species shift and resistance change by determining the frequency of fields with resistance to each herbicide.

Results

Avena spp. was found in 43% of all cropping fields surveyed and was at sufficient density to be collected from 23% of the 466 crops fields sampled (Fig. 1). The common crops were wheat (Triticum aestivum), barley (Hordeum vulgare L.) and canola (Brassica napus L.), comprising 65%, 12% and 10% of cropped fields, respectively (Owen et al. 2014). Avena was most commonly found in cereal crops, with 74% of Avena samples coming from wheat fields and 15% from barley fields. Avena spp. was distributed across the entire agricultural region (Fig. 1), but was more common in the central grain belt (zone M3) and the higher rainfall southern cropping areas (zones H4, M4). At crop maturity, no Avena plants were found within the sampling

area in 57% of fields; *Avena* spp. was difficult to find in 20% of fields; 20% of fields had weed densities <1 plant m²; and only 3% of fields had medium densities (1–10 plants m⁻²).

Resistance to ACCase-inhibiting herbicides

Screening of the *Avena* populations randomly collected from fields in the Western Australian grain belt revealed widespread resistance to the ACCase-inhibiting herbicide diclofop-methyl. Of the 128 *Avena* populations treated with diclofop-methyl, 61 populations contained resistant plants (Table 2). Of these resistant populations, only seven had ≥20% plant survival, whereas 54 populations had <20% survival (Fig. 2). The majority of resistant populations were found in the central agricultural region (zones H2, M2, L2, H3, M3, L3) where *Avena* were more prevalent; however, resistant populations were present in most of the agricultural areas (Table 3).

Resistance to other ACCase-inhibiting herbicides was markedly lower than that found for diclofop-methyl. Of the 120 populations treated with fenoxaprop, (which is widely used for wild oat control), only 32 populations contained resistant plants (Table 2). Of the resistant populations, 28 populations had <20% plant survival, whereas only four had >20% survival (Fig. 2). These resistant populations mostly came from the central agricultural region (Table 3). Resistance to tralkoxydim and sethoxydim ('dim' herbicides) was lower than that found to the 'fop' herbicides, with only 10 populations containing plants resistant to these herbicides (Table 2). These populations were confined to the medium and high rainfall zones of the central grain belt (Table 3, Fig. 2). For the similar herbicide pinoxaden, only three populations contained resistant plants and were confined to the medium rainfall zones (M3, M4) in the central grain belt (Tables 2, 3, Fig. 2).

Resistance to ALS-inhibiting herbicides

ALS-inhibiting herbicides have been widely used in the Western Australian grain belt although early ALS-inhibiting herbicides did not target *Avena* species. This survey found that of the 120 *Avena* populations screened to the ALS-inhibiting sulfonylurea herbicide mesosulfuron, only one population exhibited ≥20% plant survival to this herbicide, whereas another population had <20% survival (Table 2). Of the 101 populations treated with the imidazoline class of ALS-inhibiting herbicides (imazamox+imazapyr), only one population had ≥20% survival whereas all other populations were susceptible (Table 2). Resistance to the ALS-inhibiting herbicides was confined to the central agricultural regions (zones M3, M4) (Table 3).

Resistance to anti-microtubule mitotic disrupter herbicides

Of the 104 *Avena* populations treated with the mid-season herbicide flamprop-methyl, only eight populations contained resistant plants. Of these, five populations had \geq 20% plant survival whereas three had <20% survival (Table 2). Resistance was generally confined to the central agricultural region (Table 3) where *Avena* spp. was more prevalent (Fig. 1).

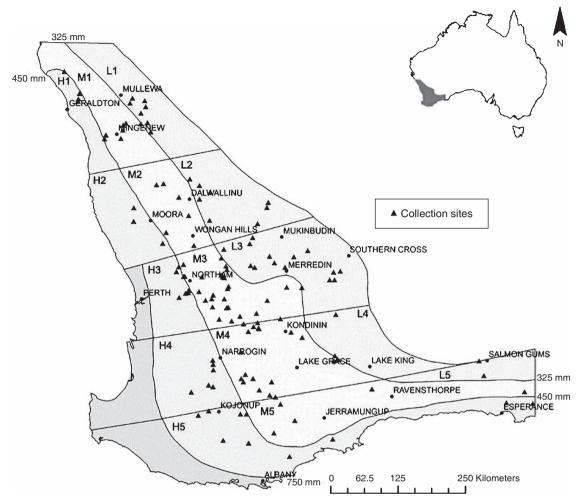


Fig. 1. Map of south-western Western Australia showing the agronomic zones of the grain belt where *Avena* populations were collected for herbicide resistance testing. Average annual rainfall isohyets are shown. Rainfall regions are represented by H (High 450–750 mm), M (Medium 325–450 mm), and L (Low <325 mm). Zones are signified by 1 (North), 2 (North central), 3 (Central), 4 (South central) 5 (South).

Table 2. The number of *Avena* spp. populations collected in 2010 in each resistance category (fully susceptible, <20% plant survival or ≥20% plant survival) for each herbicide

Herbicide	Susceptible	<20% survival	≥20% survival
Triallate	103	0	0
Diclofop	67	54	7
Fenoxaprop	88	28	4
Pinoxaden	108	1	2
Tralkoxydim	108	7	3
Sethoxydim	118	4	6
Mesosulfuron	118	1	1
Imazamox + Imazapyr	100	0	1
Glyphosate	109	0	0
Paraquat	98	0	0
Flamprop	96	3	5

Resistance to other mode-of-action herbicides

Populations were treated with the broad-spectrum herbicides glyphosate and paraquat, and the pre-emergent herbicide triallate. All populations were susceptible at Australian field rates, indicating that *Avena* populations with resistance to ACCase- or ALS-inhibiting herbicides are effectively controlled by these different herbicide modes of action.

Multiple resistance

All populations were treated independently with the herbicides used in this survey to determine the extent of multiple resistance. The number of populations with resistance to more than one herbicide mode of action was rare and confined to the central agricultural region, with only 9 of the 128 *Avena* populations having resistance across two or more herbicide modes of action (Table 4). These multi-resistant populations were predominantly from the medium and low rainfall zones of the eastern grain belt (M2, L2, L3, M3, M4).

Change in resistance levels from 2005 to 2010

Avena resistance surveys were conducted in the same region in 2005. During the 5 years between the two surveys, resistance

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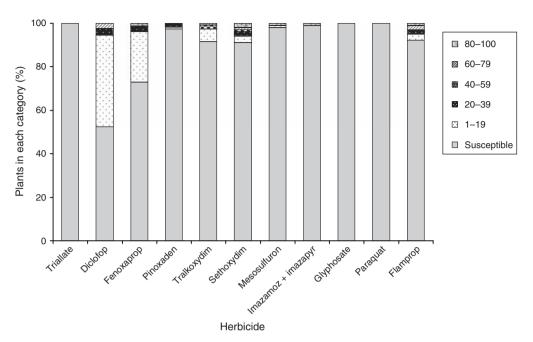


Fig. 2. Severity of resistance to each herbicide tested. Resistance intervals are 0% (all plants are susceptible), 1–19%, 20–39%, 40–59%, 60–79% and 80–100% of plants surviving each herbicide treatment.

levels to the ACCase-inhibiting herbicides have remained relatively steady (Table 5). In 2005, no resistance was detected for the ALS-inhibiting herbicides and flamprop; however, 5 years later up to 5% of populations contained plants resistant to these herbicides (Table 5).

Discussion

Although in 2010 Avena was found in many cropping fields, its frequency (43%) and distribution were similar to those recorded in the resistance survey conducted in 2005 (Owen and Powles 2009). Avena was more prevalent in the central agricultural region and in the higher rainfall zones, which is consistent with a recent survey examining changes to weed incidence in agricultural fields (Borger et al. 2012). The authors showed that the overall incidence of Avena had decreased over the 10-year survey period, from 54% in 1998 to 36% in 2008. This may be due to the range of herbicide options available for this species, less widespread herbicide resistance than in, for example, Lolium rigidum, or climatic conditions during the survey period, which affected species distribution (Borger et al. 2012).

The proportion of *Avena* populations resistant to the ACCase herbicides (48%) was lower than that of the 2005 survey (71%) although these were confined to the same agricultural zones (Owen and Powles 2009). Some of the 2005 survey populations were screened to a range of doses of ACCase-inhibiting herbicides to quantify their level of resistance, and differences in the resistance profile between populations were found (Ahmad-Hamdani *et al.* 2012). This was due to variations in their specific ACCase gene mutations (Yu *et al.* 2013) and/or to enhanced rates of herbicide metabolism (Ahmad-Hamdani *et al.* 2013). Therefore, it is likely that the ACCase-resistant populations found in the present survey may also have both

target site and non-target site resistance mechanisms, although this has not yet been investigated.

To our knowledge, this survey has detected the first ALS-inhibiting herbicide-resistant *Avena* populations from Western Australia. One population had a very high number of individuals surviving the sulfonylurea herbicide mesosulfuron and the imidazolinone herbicide mixture containing imazamox + imazapyr, whereas the other population had a lower proportion of plants resistant only to mesosulfuron. Both of these populations also had resistance to ACCase-inhibiting herbicides. Resistance to the ALS-inhibiting herbicides was not detected in surveys of *Avena* in New South Wales and Tasmania (Broster *et al.* 2011, 2013; Broster *et al.* 2012a), although it has previously been identified in *Avena* populations in other grain-growing regions of Australia and worldwide (Beckie *et al.* 1999, 2002; Heap 2015).

Resistance to the mid-season season herbicide flampropmethyl was also detected here for the first time in a random survey of *Avena* in Western Australia. In total, eight populations had varying levels of resistance to flamprop. All of these populations had resistance to a range of ACCase-inhibiting herbicides, except that most were susceptible to pinoxaden. Only one of the flamprop-resistant populations had resistance to the ALS-inhibiting herbicides. A recent survey in New South Wales found a similar proportion (10%) of *Avena* populations were resistant to flamprop (Broster *et al.* 2011), and flampropresistant populations have previously been identified in this region through commercial failure (Broster 2004). Surveys in Tasmania found no resistance in *Avena* spp. (Broster *et al.* 2012*a*), although flamprop resistance is known in other areas (Heap 2015).

ACCase- and ALS-inhibiting herbicide resistance is widespread in the cross-pollinated grass weed species *Lolium*

The percentage of Avena spp. populations in the high-resistance (\(\inftit{\gamma}\)20% surviving plants (H)), low-resistance (\(\inftit{\gamma}\)0% surviving plants (L)) or fully susceptible (0% surviving plants (S)) categories by agronomic zone (refer to Fig. 1) to herbicides tested in this survey Fable 3.

No. Diclosop Fenoxaprop nonulations	Diclofop Fenoxaprop	Diclofop Fenoxaprop	op Fenoxaprop	Fenoxaprop	noxaprop	rop	Pin	Pinoxaden	и	Tra	Tralkoxydim	lim	Se	Sethoxydim	dim	Me	sosul	Mesosulfuron		Intervix	νix	FI	Flamprop	dı
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50 50 0 50	50 50 0 50	50 0 50	0 50			50	0	0	100	0	25	75	0	0	100	0	0	100	0	0	100	0	0	100
0	60 20 0 60	20 0 60	09 0			40	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	20	8
38 50 0	38 50 0 62	50 0 62	0 62			38	0	0	100	12	0	88	13	12	75	0	0	100	0	0	100	14	0	86
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0 29	67 0 17	67 0 17	0 17			83	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
53 6 22	53 6 22	53 6 22	6 22			72	0	7	93	0	9	94	S	0	95	9	0	95	7	0	93	0	0	100
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60 0 10	60 0 10	60 0 10	0 10	0 10 90	10 90	06	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
5 0 40 60 0 100	0 0 09	0 0 09	0 0	0 0 100	0 100	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100
2 0 0 100 0 0 100	0 0	0 0	0 0	0 0 100	0 100	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100	0	0	100

Table 4. The number of *Avena* populations with multiple resistance to different herbicide groups

Herbicide combination	No. of populations with resistant plants
All herbicides tested	0
Acetyl-Co A carboxylase (ACCase)+	1
Acetolactate synthase (ALS) + Flamprop	
ACCase + ALS	2
ACCase + Flamprop	8
ALS + Flamprop	2

rigidum in Australian cropping fields (Boutsalis et al. 2012a; Broster et al. 2013; Owen et al. 2014) whereas for self-pollinated grass weeds such as Bromus, Hordeum and Avena, resistance to these herbicides, particularly the ALS inhibitors, is rare (Owen and Powles 2009; Broster et al. 2010, 2011, 2012a; Boutsalis et al. 2012b; Owen et al. 2015; present study). This could be because the self-pollination syndrome of Avena, Bromus and Hordeum species means that the transfer of resistance genes is generally slower and less widespread. The particular herbicide selection intensity may also slow resistance dispersion, as some herbicides only target a specific species whereas others cover a broad range of grass species. The complicated inheritance of resistance alleles in hexaploid species such as Avena also means that resistant individuals could have one, two or three different gene mutations (Yu et al. 2013). The level of resistance endowed by these target site mutations in hexaploid species is usually lower due to the dilution of resistance alleles compared with diploid species such as Lolium (Han et al. 2015).

A potential method for controlling herbicide-resistant Avena populations is to target the seed bank, but the characteristics of this genus make it less amenable to harvest weed seed control than some of the other major weeds. Avena spp. often emerge throughout the growing season due to seed dormancy, which contributes to the persistence of the species by allowing moredormant individuals to avoid in-crop herbicide applications by germinating later. The seed bank is also replenished annually because Avena seeds tend to mature earlier than the crop species they infest, meaning their seed can shatter readily before or at crop harvest, with seed shed varying from 44% to 95% (as cited in Shirtliffe et al. 2000; Beckie et al. 2012). Other recent work suggests that up to 84% of Avena seeds remain on the plant at wheat maturity, but that high seed shed (up to 39%) takes place in the 28 days after crop maturity (Walsh and Powles 2014). Therefore, unless all crops can be harvested when the crop first matures, there is likely to be substantial Avena seed dispersal before harvest. Seed retention in Avena species was also significantly affected by seasonal weather conditions (Shirtliffe et al. 2000; Walsh and Powles 2014), genetic differences, and emergence timing of the parent plants (Shirtliffe et al. 2000). This reduces the number of options available to remove seed from the cropping system during harvest; and therefore, there is often high reliance on early post-emergent, crop-selective herbicides to control Avena in crop fields.

The control of *Avena* populations with herbicides alone becomes increasingly difficult when multiple resistance

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Herbicide	Susceptible	2005 <20% survival	≥20% survival	Susceptible	2010 <20% survival	≥20% survival
Diclofop	29	55	16	52	42	6
Fenoxaprop	78	17	5	73	24	3
Pinoxaden	97	1.5	1.5	97	1	2
Tralkoxydim	95	5	0	91	6	3
Sethoxydim	76	12	12	92	3	5
Mesosulfuron	100	0	0	98	1	1
Imazamoz + Imazapyr	100	0	0	99	0	1
Flamprop	100	0	0	92	3	5

Table 5. The change in herbicide resistance status between 2005 and 2010 for *Avena* populations collected from the same regions of the Western Australian grain belt (values are the percentage of total populations tested)

mechanisms exist in some populations, combined with the complicated inheritance of resistance alleles in Avena spp. (Yu et al. 2013). Often, control techniques rely on agronomic management by delaying sowing to maximise weed emergence and using effective broad-spectrum herbicides; using competitive crops to reduce weed vigour; crop topping using late season herbicide application; and combining these with techniques which target weed seeds at harvest. The latter include burning, baling, using chaff carts, (which have also been shown to reduce seed dispersal) (Shirtliffe and Entz 2005) and using the Harrington Seed Destructor, which has shown high destruction (>90%) of Avena seed (Walsh et al. 2012). However, these techniques rely on high seed retention to capture the weed seeds during the harvest operation. All techniques which prevent the seeds returning to the soil seed bank, whether chemical, management-based or mechanical, are required to reduce the weed burden in the following years to maintain the sustainability of cropping systems in the future.

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