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A Review of Herbicide Resistance in Iran

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Continuous use of herbicides has triggered a phenomenon called herbicide resistance. Nowadays, herbicide resistance is a worldwide problem that threatens sustainable agriculture. A study of over a decade on herbicides in Iran has revealed that herbicide resistance has been occurring since 2004 in some weed species. Almost all the results of these studies have been published in national scientific journals and in conference proceedings on the subject. In the current review, studies on herbicide resistance in Iran were included to provide a perspective of developing weed resistance to herbicides for international scientists. More than 70% of arable land in Iran is given over to cultivation of wheat, barley, and rice; wheat alone covers nearly 52%. Within the past 40 years, 108 herbicides from different groups of modes of action have been registered in Iran, of which 28 are for the selective control of weeds in wheat and barley. Major resistance to ACCase-inhibiting herbicides has been shown in some weed species, such as winter wild oat, wild oat, littleseed canarygrass, hood canarygrass, and rigid ryegrass. With respect to the broad area of wheat crop production and continuous use of herbicides with the sole mechanism of action of ACCase inhibition, the provinces of West Azerbaijan, Tehran, Khorasan, Isfahan, Markazi, and Semnan are at risk of resistance development. In addition, because of continuous long-term use of tribenuron-methyl, resistance in broadleaf species is also being developed. Evidence has recently shown resistance of turnipweed and wild mustard populations to this herbicide. Stable monitoring of fields in doubtful areas and providing good education and training for technicians and farmers to practice integrated methods would help to prevent or delay the development of resistance to herbicides.
Nomenclature: Haloxyfop-r-methyl ester; tribenuron methyl; hood canarygrass, *Phalaris paradoxa* L. PHAPA; littleseed canarygrass, *Phalaris minor* Retz. PHAMI; rigid ryegrass, *Lolium rigidum* Gaud. LOLRI; turnipweed, Rapistrum rugosum (L.) All. RASRU; wild mustard, *Sinapis arvensis* L. SINAR; wild oat, *Avena fatua* L. AVEFA; winter wild oat, *Avena ludoviciana* (Durieu) AVELU.
Key words: ACCase inhibitor, ALS inhibitor, herbicide resistance, PSII inhibitor, weed.

Among the chemicals that are currently used in agricultural systems, special attention is devoted to herbicides. They are the most frequently used inputs supported by subsidy on the farms (Vila-Aiub et al. 2008). Increased crop yields have relied more and more on herbicide use. During the last two decades, new herbicides with broader spectra of control have been introduced. However, although herbicides have been beneficial to agricultural production, negative consequences, including suspected environmental impacts, have raised concerns about further chemical use. The increased use of herbicides has resulted in resistance—the most important negative side effect. Natural weed populations probably contain individual plants (biotypes) that are resistant to herbicides, irrespective of the mechanism of action of the herbicides applied. Continuous use of herbicides with same mode of action will expose the weed population to a selection pressure that leads to an increase in the number of surviving resistant individuals. As a consequence, the resistant weed population may increase to the point that adequate weed control cannot be achieved by the application of those herbicides (Heap 2016).

In 1964 the first case of herbicide resistance in weeds was identified; since then 464 biotypes from 249 weed species have shown resistance in worldwide populations, 144 of which are dicotyledonous and 105 monocotyledonous. Herbicide resistance was found in 22 of 25 known herbicide sites of action, including 157 herbicides. The reports of resistant weed biotypes are from 66 countries (Heap 2016).

According to the Koeppen-Geiger classification (Kottek et al. 2006), Iran has four zones, temperate to Mediterranean and subtropical/tropical, with huge deserts and major problems with water

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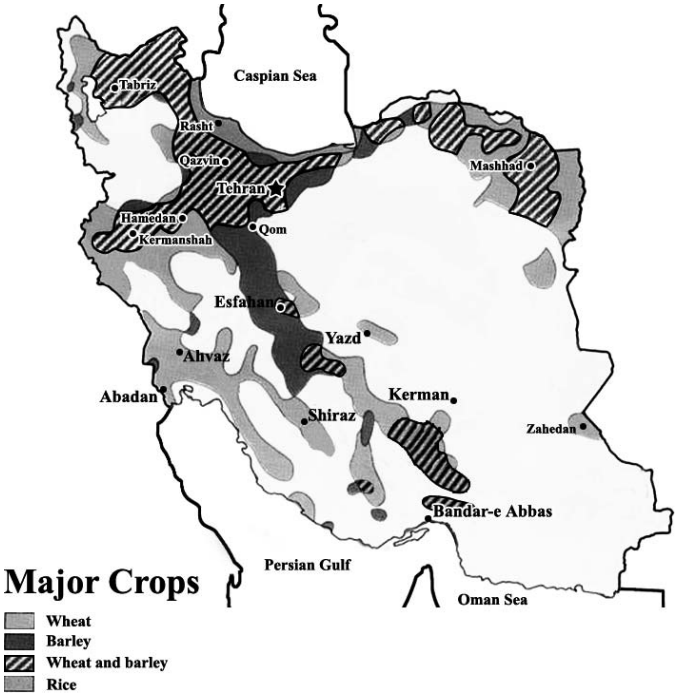

Figure 1. Map of Iran's major crops.

shortages. The arid regions have experienced increasing water shortage problems, which has limited the use of some effective cultural methods, such as tillage, crop rotation, and stale seed bed, resulting in increasing reliance on chemical control. It has been more than 40 yr since the first herbicide registration in Iran. Currently, over 100 herbicides are used in Iranian fields, mostly with PSII inhibitors, ACCase and ALS inhibitors, fatty acid biosynthesis inhibitors, and hormones (Zand et al. 2007). ACCase and ALS inhibitors and their mix have become the dominant herbicides in recent years. However, weed resistance to herbicides caused some drawbacks in efficiency of chemical control of weeds (Zand et al. 2007). Studies on herbicide resistance in Iran commenced in 2001 with reports of developing resistance to ACCase-inhibiting herbicides (Zand et al. 2004).

Strategies of weed management are highly affected by herbicide-resistance issues. Resistant weeds in Iranian fields have been described by several articles published in national journals. This rising issue has to be taken seriously in decision-making programs. The current review compiles information obtained through experiments or surveys on the subject conducted during recent years. Almost all the results of these studies have been published in national scientific journals and in proceedings of conferences on the subject. In the current review, studies on herbicide resistance in Iran were included to provide a perspective of developing weed resistance to herbicides for international scientists. More than 70% of arable land in Iran is given over to cultivation of wheat, barley, and rice; wheat alone covers nearly 52%. Besides sharing experience, partially specific to Iranian cropping systems, this review should help improve bugs in management programs that lead to increasing issues of weed resistance to herbicides.

Table 1. Area of wheat fields of Iran infested by weeds resistant to ACCase inhibitors.

Province	Approximate level (ha)
Khuzestan	160,000
Fars	80,000
Kermanshah	2,000
Esfahan	2,000
Ilam	1,500
Golestan	1,500
Tehran	500
Semnan	500
Chahar-Mahal and Bakhtiyari	200

Resistance to ACCase Inhibitors

Since the first study to detect weed biotypes resistant to herbicides, conducted in 1997 (Zand et al. 2004), several biotypes of littleseed canarygrass, hood canarygrass, wild oat, sterile oat (*Avena sterilis* L.), and rigid ryegrass have shown resistance to ACCase inhibitors. These detection reports of resistant weeds were entirely attributed to winter cereals (mostly wheat [*Triticum aestivum* L.] and barley [*Hordeum vulgare* L.]) in central Iran (Minbashi Moeini et al. 2008). Of the 12.96 million ha of Iranian agricultural areas, 9.37 million ha (about 72.28%) are in cereals (73.42% wheat and 16.73% barley; Anonymous 2012) (Figure 1). Chemical control is the major method for weed control in 30% of the fields. Fifty-nine percent of the fields are treated for controlling broadleaf weeds, 32% are treated by graminicides, and dual-purpose herbicides are applied in the remaining fields (Anonymous 2012). The total amount of herbicides applied in cereals has doubled during the past 25 yr (Zand and Baghestani 2008).

Studies in Khuzestan, Fars, Kermanshah, Ilam, Golestan, Tehran, and Semnan have shown that weed resistance to herbicides, especially to the ACCase inhibitors, has increased in wheat fields with increased herbicide application rate (Table 1).

Wheat yield suffered a loss of 25 to 50% on average due to weed competition in semiarid

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environments (Beckie 2007). Wheat is the most strategic crop in Iran, and weeds caused a 23% reduction in wheat yield annually (Gherekhloo et al. 2010a). Nonchemical methods do not meet requirements for weed control; therefore, herbicide application is necessary. So far, 27 herbicides consisting of 11 graminicides, 10 broadleaf killers, and 7 dual-purpose herbicides have been registered for weed control in wheat, which makes up a large proportion of registered herbicides in Iran (Table 2). Among grass herbicides, benzoylprop ethyl, triallate, and trichloroacetate sodium are no longer approved for use in wheat. Also, two herbicides, difenzoquat and flamprop-M-isopropyl, were found to be inefficient for weed control in wheat. The remaining ones are acetyl co-enzyme A carboxylase inhibitors. Therefore, this high-risk herbicide family has been the sole option of chemical control for many years. In summary, weed control in a large area of cereal cropping has relied on chemical control by herbicides with the same mode of action. For example, diclofop-methyl has been a frequently used herbicide since 1980. Long-term, continuous use of this herbicide raised concerns that led to prototype studies on resistance occurrence in 1998. Results suggested no weed resistance to diclofop methyl. The history of herbicide applications during the 1990s shows that applied herbicides had several mechanisms of action. Difenzoquat, benzoylprop ethyl, and diclofop methyl (in the 1970s and 1980s), and the herbicides fenoxaprop-p ethyl (1993), clodinafop propargyl (1994), and tralkoxydim (1997) provided diversity in herbicides that caused delay in weed resistance development. Shortly thereafter, in 2000 studies showed developing resistance of littleseed canarygrass, hood canarygrass, wild oat, winter wild oat, and rigid ryegrass to ACCase inhibitors in wheat fields of Fars, Golestan, Isfahan, Lorestan, Ilam, and Khuzestan. Zand and Baghestani (2008) suggested that removing herbicides such as difenzoquat and benzoylprop ethyl from the herbicide application programs led to a continuous application of diclofop methyl, fenoxaprop-p ethyl, and clodinafop propargyl which have the same mechanism of action.

The discriminating dose was used to compare the rate of herbicide resistance in various species. It is the minimum dose required to kill susceptible plants (Beckie et al. 2000). Weed species show different levels of resistance based on the discriminating dose measurement. Elahifard (2005), based on a 50% reduction in seedling populations, suggested that the discriminating dose of fenoxaprop-p ethyl and diclofop methyl for littleseed canarygrass populations would respectively be 0.8 and 0.4 ppm a.i. Using the same criterion, Benakashani et al. (2006) determined the discriminating doses of diclofop methyl, fenoxaprop-p ethyl, and clodinafop propargyl for winter wild oat populations to be 0.01, 4, and 0.1 ppm a.i., respectively. In comparison, on the basis of 80% reduction in seedling growth, Rastgoo (2007) and Gherekhloo (2008) estimated the discriminating doses for the respective herbicides at 10, 0.6, and 0.08 for winter wild oat and at 8.05, 1.05, and 0.093 ppm a.i. for littleseed canarygrass, respectively. Table 3 shows cross-resistance development in most studied populations. Elahifard (2005) reported cross resistance in the littleseed canarygrass populations to diclofop methyl and fenoxaprop-p ethyl. Cross resistance was also shown in winter wild oat populations to diclofop methyl, fenoxaprop-p ethyl, and clodinafop propargyl (Rastgoo 2006; Zand et al. 2009). There were also biotypes of winter wild oat that showed a wider cross resistance to the herbicides diclofop methyl, fenoxaprop-p ethyl, clodinafop propargyl, tralkoxydim, and pinoxaden (Benakashani et al. 2010; Gherekhloo 2008; Gherekhloo et al. 2012). Some biotypes of winter wild oat collected from Fars showed cross resistance also to clodinafop propargyl, sethoxydim, and pinoxaden (Benakashani et al. 2010). Esmailzadeh et al. (2012) found four pinoxaden-resistant biotypes of rigid ryegrass from Fars. There were also some levels of resistance to clodinafop propargyl, pinoxaden, and their mixture (pinoxaden + clodinafop propargyl) in the winter wild oat populations collected from Khuzestan. However, the level of resistance to pinoxaden + clodinafop propargyl was lower than the levels shown to the other herbicides (Najafi et al. 2012).

Mechanism of Resistance to ACCase Inhibitors

Initial studies to evaluate resistance development are based on plants' ability to survive herbicide application; therefore, complementary studies are required to find out the physiological or molecular basis of resistance (Uludag et al. 2007). Knowledge about the mechanism of resistance to herbicides is necessary to decision making for weed control in infested areas. For example, if the mechanism of action of resistance is altering the site of action, applying herbicides with different mechanism of action could achievably break the resistance—

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Table 2. List of registered herbicide for wheat fields of Iran

HRAC group (WSSA)[a]	Chemical family	Common name	Commercial name	Formulation	Mode of action
Graminicides					
A (1)	Aryloxyphenoxy propionate	Diclofop-methyl	Illoxan	EC 36%	ACCase inhibitor
		Fenoxaprop-p-ethyl	Puma super	EW 7.5%	
		Clodinafop propargyl	Topik	EC 8%	
	Cyclohexandione	Tralkoxydim	Grasp	EC 10%	
	Phenylpyrazoline	Pinoxaden	Axial	EC 4.5%	
	Phenylpyrazoline + aryloxyphenoxy propionate	Pinoxaden + clodinafop propargyl	Traxus	EC 4.5%	
N (8)	Thiocarbamate	Triallate	Avadex BW	EC 46%	Inhibition of lipid synthesis
Z (26)	Pyrazolium	Difenzoquat methyl sulfate	Avenge	SL 25%	Unknown
Z (25)	Arylaminopropionic acid	Flamprop-M-isopropyl	Suffix BW	EC 20%	Unknown
Z (?)	Dichloro aniliopropionate	Sodium trichloroacetate	Orbitax T95G	GR 95%	Unknown
Z (?)	Dichloro aniliopropionate	Benzoylprop ethyl	Suffix	EC 20%	Unknown
Broadleaf killers					
O (4)	Phenoxy-carboxylic-acid	2,4-D	U-46 Difluid	SL 67.5%	Synthetic auxins
		2,4-D + MCPA	U-46 cambifluid	SL 72%	
		Mecoprop + dichlorprop + MCPA	Duplosan super	SL 60%	
	Phenoxy-carboxylic-acid + benzoic acid	2,4-D + dicamba	Dialan super	SL (34.4 + 12)%	
	Benzoic acid	Dicamba	Banvel K	EC 49%	
B (2)	Sulfonylurea	Tribenuron methyl	Granstar	DF 75%	ALS inhibitor
C3 (6)	Nitrile	Bromoxynil	Pardner	SL 22.5%	PSII inhibitor
C3 (6) + O (4)	Nitrile + phenoxy-carboxylic-acid	Bromoxynil + MCPA	Bromicide MA	EC 49%	PSII inhibitor + synthetic auxins
B (2) + C1	Sulfonylurea + triazine	Triasulfuron + terbutryne	Logran extra	WG (60+4)%	ALS inhibitor + PSII inhibitor
B (2) + O (4)	Sulfonylurea + benzoic acid	Triasulfuron + dicamba	Lentour	WG 70%	ALS inhibitor + synthetic auxins
Dual-purpose herbicides					
B (2)	Sulfonylurea	Imazamethabenz-methyl	Assert	EC 25%	ALS inhibitor
		sulfosulfuron	Apyrus	DF 75%	
		Mesosulfuron-methyl + iodosulfuron + mefenpyr	Chevalier	WG 6%	
		Mesosulfuron-methyl + iodosulfuron + mefenpyr	Atlantis	OD 1.2%	
		Metsulfuron + sulfosulfuron	Total	WG (75 + 15)%	
C2 (7) + F1 (12)	Urea + pyridinecarboxamides	Isoproturon + diflufenican	Panther	EC 55%	PSII inhibitor + inhibition of carotenoid biosynthesis at the phytoene desaturase step
C2 (7)	Urea	Methabenzthiazuron	Tribunil	WP 70%	PSII inhibitor

[a] Abbreviations: HRAC, Herbicide Resistance Action Committee; WSSA, Weed Science Society of America.

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Table 3. Summary of some studies on herbicide-resistant weeds in Iran.

Weed species	Location	Crop	Herbicide	Recommended rate	Criterion	Degree of resistance		Herbicide group	Mechanism of resistance	Reference	Report/ citation year
						Petri dish	Greenhouse				
				ppm							
Phalaris minor	Golestan	Wheat	Diclofop-methyl	0.4	EC_{50}	1.57–4.05	7.26–24.25	ACCase inhibitors	Unknown	Elahi Fard and Zand	2005
			Fenoxaprop-p-ethyl	0.8	EC_{50}	1.71–3.64	9.95–24.3				
P. minor	Fars	Wheat	Diclofop-methyl	8.05	EC_{80}	1.3–9.71	1.79–11.78	ACCase inhibitors	Target based	Gherekhloo	2008
			Fenoxaprop-p-ethyl	1.05	EC_{80}	1.56–41.02	1.71–11.66				
			Clodinafop propargyl	0.093	EC_{80}	12.53–69.95	7.65–8.13				
Phalaris paradoxa	Fars	Wheat	Diclofop-methyl	—	—	—	—	ACCase inhibitors	Unknown	Zand	2007
Avena ludoviciana	Khuzestan	Wheat	Diclofop-methyl	10	EC_{80}	1.2–2.9	4.99–7.28	ACCase inhibitors	Target based	Rastgoo	2006
			Fenoxaprop-p-ethyl	0.6	EC_{80}	1.36–2.16	2.85–6.8				
			Clodinafop propargyl	0.08	EC_{80}	1.74–4.12	2.26–6.37				
A. ludoviciana	Khuzestan	Wheat	Clodinafop propargyl	0.01	EC_{50}	1.25–4.2	13.73–22.66	ACCase inhibitors	Unknown	Benakashani et al.	2006
Avena fatua	Fars	Wheat	Diclofop-methyl	—	—	—	—	ACCase inhibitors	Unknown	Zand et al.	2004
			Clodinafop propargyl	—	—	—	—				
A. ludoviciana	Fars	Wheat	Clodinafop propargyl	0.08	EC_{50}	4.06–26.26	1.53–34.07	ACCase inhibitors	Unknown	Sasanfar et al.	2009
			Pinoxaden	0.08	EC_{50}	1.83–32.21	2.27–26.89				
			Sethoxydim	1.32	EC_{50}	1.19–15.70	1.17–12.16				
Lolium rigidum	Fars	Wheat	Diclofop-methyl	—	—	—	—	ACCase inhibitors	Unknown	Zand et al.	2008
			Clodinafop propargyl	0.2	EC_{50}	1.64–13.55	299.6–2999.9		Target based	Dastoori and Zand	2010
L. rigidum	Fars	Wheat	Clodinafop propargyl	—	—	—	1.69–73.46	ACCase inhibitors	Unknown	Zand et al.	2009
A. ludoviciana	Khuzestan	Wheat	Mezosulfuron + iodosulfuron	0.057	EC_{50}	2.33–3.00	4.20–17.79	ALS	Unknown	Aghajani et al.	2009

although, for herbicide resistance on the basis of increased metabolism, the solution would be more complicated to achieve (Holt et al. 1993).

Study on 14 populations of littleseed canarygrass collected from Fars showed an insensitive ACCase enzyme that presented a significantly higher resistance to fenoxaprop-p ethyl (resistance factor > 8), diclofop methyl (resistance factor > 5) and clodinafop propargyl (resistance factor > 7). The other populations with lower resistance levels had a similar moderate sensitivity of the ACCase enzyme to herbicides (Gherekhloo et al. 2011).

Molecular Basis of Resistance to ACCase

Mutations in the gene encoding ACCase commonly results in an altered enzyme, which may cause resistance to inhibitors of ACCase (Yu et al. 2007). Gene extraction asserted that the mechanism of resistance is an altered target site. Seven mutations have so far been found in the plastid gene encoding ACCase enzyme in the range of carboxyltransferase. The mutations all led to alterations in the ACCase structure resulting in resistance to herbicide (Liu et al. 2007). Rastgoo et al. (2006) and Dastoori et al. (2010), respectively, studied the molecular basis of the resistance of winter wild oat and rigid ryegrass populations, with the use of the dCAPS method. Rastgoo et al. (2006) showed resistance occurrence in 19 populations of winter wild oat, from which 10 populations were ACCase-1781 mutant. Dastoori et al. (2010) have also reported consistent results in rigid ryegrass. Resistance due to substitution in isoleucine-1781-leucine leads to cross resistance to both the APP and CHD herbicides excluding clethodim (Yu et al. 2007). Rastgoo (2006) and Dastoori et al. (2010) suggested that the mechanism of resistance in the studied populations did not follow any alteration in the target site. Another study (which used the dCAPS method) on the littleseed canarygrass populations collected from Fars indicated that there was no mutation in ACCase 1781 (Gherekhloo 2008). However, a bioassay test confirmed that an altered enzyme resulted in resistance to the ACCase inhibitors. To determine the mutations responsible for increased resistance, a molecular study was conducted that showed mutations at codons 2027 or 2028 or both in three populations (Gherekhloo et al. 2012). Liu et al. (2007) suggested that mutation at tryptophan-2027-cyctein leads to resistance to APP herbicides. Also, change in asparagine-2078-glycine causes resistance to APP and CHD herbicides including clethodim. Therefore, it is predicted that mutations at codons 2027 and 2078 lead to resistance to many herbicides of the APP and APP + CHD groups, respectively. Studies on the littleseed canarygrass populations resistant to diclofop methyl, fenoxaprop-p ethyl, and clodinafop propargyl indicated that the ACCase-2078 mutants that were never sprayed with tralkoxydim and pinoxaden could develop resistance to these herbicides (Gherekhloo and Derakhshan 2012).

Herbicide metabolism and reduction in absorption and translocation of herbicides are also mentioned as important mechanisms for herbicide resistance (Volenberg and Stoltenberg 2002). However, these mechanisms lead to lower resistance rates. Studies on absorption and translocation of diclofop methyl in the littleseed canarygrass populations showed no significant difference between resistant and sensitive populations (Gherekhloo et al. 2010b). Eight of 17 biotypes of winter wild oat suspected to be resistant to clodinafop propargyl were shown to have mutation of isoleucine-1781-leucine in plastidic ACCase enzyme. No mutation of isoleucine-2041-asparagine leading to any resistance was observed in populations. For the nine populations that were shown to be resistant, the molecular mechanism of resistance is unknown; however, it may be explained through the mutation in another location or a metabolism-based mechanism (Zand et al. 2009).

Resistance to ACCase in Other Crops

After long-term application of herbicides haloxyfop ethoxy ethyl (from 1985), fluazifop-p butyl (from 1985), quizalofop ethyl (from 1988), sethoxydim (from 1985), and haloxyfop-R methyl ester (from 2000) in Iranian sugar beet (*Beta vulgaris* L.) and canola (*Brassica napus* L.) farms, still no evidence of resistance to these herbicides was found in sugar beet cropping. The prevalent application of nonchemical methods in integration with chemical control is mentioned as efficient management tool that prevented the resistance development in sugar beet farms. In contrast in canola cropping, studies showed resistance to haloxyfop-R methyl ester in the winter wild oat populations collected from Golestan (Gherekhloo, unpublished data).

Haloxyfop-R methyl has been the single herbicide option available for grass weeds control in canola during the past 10 yr. In Golestan with more than 18,000 ha of canola cropping (Anonymous 2012),

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winter wild oat is recognized as the most important grass weed. The continuous use of haloxyfop-R methyl and lack of an integrated method for weed control in canola farms are the reasons given for resistance development to haloxyfop-R methyl in the winter wild oat populations. Wheat or canola as winter cropping followed by a summer crop is the prevalent crop rotation applied in the Golestan farms. However, different graminicides belong to ACCase-inhibiting herbicides are used for the control of winter wild oat in wheat and canola, the herbicides applied almost belong to same family, i.e., ACCase inhibitors. Therefore, this crop rotation does not deliver diversity in the mechanism of action of herbicides to prevent resistance development.

Resistance to ALS Inhibitors

There are also reports on resistance development to the ALS-inhibiting herbicides in Iran. Aghajani et al. (2009) showed resistance to the mix of mezosulfuron + idosulfuronin in some biotypes of winter wild oat in Khuzestan. The ALS inhibitors were registered in Iran to provide diversity in the mechanism of action of herbicides. However, they are also classified in the group of high-resistance risk herbicides (Beckie 2007). Tribenuron-methyl (registered in 1990) as an ALS inhibitors has been used for many years, and recently some resistance rates to this herbicide were shown in the biotypes of wild mustard and turnipweed in Golestan (Derakhsahn and Gherekhloo 2012; Najjari Kalantari 2013; Zand et al. 2012), Fars, and Khuzestan (Zand et al. 2012). Imazamethabenz-methyl (registered in 1995) is another ALS inhibitor that has been long used for weed control in wheat fields; however, no report on weed resistance to this herbicide has been so far received.

Bensulfuron methyl (1997) and sinosulfuron (1996) are also used frequently in rice fields. Field evaluations showed some tolerance to these herbicides; however, because of hand weeding commonly applied once or twice during the season, the individual resistant biotypes are mostly removed and the chance of establishing resistant colonies is severely decreased.

Resistance to PSII Inhibitors

PSII inhibitors are among the first herbicide groups imported to Iran and have a long history of application in farms. They are classified as intermediate resistance risk, and the chance of resistance development will highly increase after 11 yr of continuous usage (Beckie 2007). However, some PSII inhibitors, such as atrazine, chloridazon, and propanil, with a history of 40 yr of application in sugar beet fields, have not been reported to cause resistance in weeds (Partovi et al. 2005; Zand et al. 2004). Also in the rice fields of northern Iran and Isfahan, which have continuously received a high amount of propanil for many years, no evidence of resistance development in the barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.] populations was obtained (Bitarafan et al. 2012).

Seven out of 13 herbicides that are registered for weed control in sugar cane are PSII inhibitors (Zand et al. 2007). Sugarcane (*Saccharum officinale* L.) has been monocropped in Khuzestan for over three decades. Continuous and intensive application of PSII inhibitors has increased the probability of resistance development. Elahifard et al. (2013) reported resistance of junglerice [*Echinochloa colona* (L.) Link.] populations to atrazine in sugarcane fields of Khuzestan. DNA sequence analysis of the psbA gene, which is the target site of PSII-inhibiting herbicides, revealed that two nucleotide changes (A to G) at positions 232 and 286 conferred two amino acid substitutions from serine to glycine at residue 264 in the resistant biotypes of junglerice. A complementary test indicated the cross-resistance development in the junglerice populations to ametryn and metribuzin (Elahifard et al. 2013).

Resistance to Other Herbicide Groups

Studies in 1998 and 1999 evaluated the resistance development in the fields with a long history of herbicide applications from Groups A (ACCase inhibitors), B (ALS inhibitors), K (cell-division inhibitors), and O (synthetic auxin herbicides). However, some reports stated poor herbicide effects on weeds, results showed no resistant weed in the study farms. Bitarafan et al. (2012) evaluated resistance to butachlor, molinate, and thiobencarb in 37 suspected populations of barnayrdgrass collected from rice fields of Gillan, Mazandaran, and Fars. However, the respective herbicides were continuously used in the rice fields; no evidence of resistance development was obtained.

2,4-D + MCPA has also been used for many successive years for the control of broadleaf weeds in wheat fields. Fourteen populations of wild mustard collected from Fars, Golestan, and Khuzestan, and two populations of Indian mustard [*Brassica juncea*

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Table 4. Registered and common herbicides for control of grass weeds in wheat fields of Iran.

Trade name	Common name	Mode of action
Panther SC (50 + 5%)	isoproturon + diflufenican	PSII inhibitor + inhibition of carotenoid biosynthesis
Assert EC 25%	Imazametabenz	ALS inhibitor
Apyrus 75% WG	Sulfosulfuron	ALS inhibitor
Chevalier WG (3 + 3%)	Mezosulfuron + idosulfuron	ALS inhibitor
Illoxan EC 36%	Diclofop methyl	ACCase inhibitor
Puma super EW 7.5%	Fenoxaprop-p-ethyl + mefenpyr-diethyl	ACCase inhibitor
Topik EC 8%	Clodinafop propargyl	ACCase inhibitor
Grasp SC 25%	Tralkoxydim	ACCase inhibitor
Axial EC 10%	Pinoxaden	ACCase inhibitor

(L.) Czern.] from Fars were evaluated for the resistance to 2,4-D + MCPA. Results showed no resistance development to 2,4-D + MCPA (Zand et al. 2012).

Management of Herbicide Resistant Weeds in Wheat

Herbicides are actually a selection pressure that removes sensitive plants and provides more growth for the resistant individuals. Therefore, any attempt to reducing the selection pressure rate would delay resistance evolution. Creating diversity in herbicide programs and reinforcing the integrated methods (cultural or mechanical + chemical) can decrease the reliance on herbicides with the same mode of action (Beckie 2007; Vencill et al. 2015). For social or political reasons, rotating other crops with wheat is not possible. Therefore, herbicide rotations or mixtures are considered superior alternatives to resistant weeds management. ACCase and ALS inhibiting herbicides, and isoproturon + diflufenican are the registered herbicides for weed control in wheat (Table 4). Therefore with the available herbicides for weed control in wheat, creating diversity in herbicide mechanism of action is useful for resistance management.

Several reports on resistance development to the ACCase-inhibiting herbicides urge caution in using this herbicide group. Including other available herbicide groups in herbicide programs is recommended. However, herbicide rotation can be helpful if the mechanism of resistance is based on altered target site. For other mechanisms of resistance, including enhancing herbicide metabolism, weeds are commonly resistant to a wider range of herbicide, so management solutions would be more complicated (Beckie 2007). This indicates the importance of knowing the mechanism of resistance being developed in weed populations.

Crop Rotation

We are currently receiving many reports submitted on resistant weeds from Khuzestan province (Zand, personal communication). The reason for this might be explained in the changes undergone by crops in the rotations. For many years, wheat–sugar beet was the prevalent rotation in this region. Sugar beet is planted as a winter crop in Khuzestan because of the very hot summers. However, for several reasons, sugar cane, which is a perennial crop, has gradually been replacing sugar beet as a source of sugar. With sugar beet removed from rotations, wheat became the prevalent winter crop, followed by corn (*Zea mays* L.) that planted in early spring after harvesting wheat. In the view of weed management, wheat–corn is not an efficient crop rotation, because they are grown in different seasons and therefore have different weed flora. For example, weeds that are problematic in corn production, such as redroot pigweed (*Amaranthus retroflexus* L.), common cocklebur *(Xanthium strumarium* L.), and barnyardgrass, are not present in wheat because of their different temperature requirements for germination and growth. Similarly, the main weed species in wheat have no significant presence during corn growth. Therefore, replacing sugar beet with sugar cane and using corn in the rotation results in a monoculture system, which definitely lacks the advantages of crop rotation.

No-tillage is increasingly considered to be the preferred approach. Weed control in no-till systems is highly dependent on herbicide application. The increase in the herbicide application rates through the fields, the lack of diversity in the herbicides' mechanism of action, and longer persistence of herbicide in the soil because of plant residue remaining on the ground, are the main reasons for developing weed resistance in the area.

Conclusions

Over a decade of research on weed resistance to herbicides has revealed the development of resistance to ACCase inhibitors in wild oat biotypes (wild oat and winter wild oat) in a large area of Iran, including Golestan, Ilam, Kermanshah, Khuzestan, Fars, and Chahar Mahal-e-Bakhtiari. Other major grass weeds, such as littleseed canarygrass, hood canarygrass, and rigid ryegrass, have also shown some levels of resistance to herbicides, but in more limited areas.

Among weed populations collected from different farms, there are clear differences in resistance rates (Table 1). As a valid history of herbicide usage in fields is not available, the difference between populations cannot be properly explained. However, it is generally confirmed that current weed control methods have led to the development of resistance to herbicides in weed populations. For some herbicide families, such as ACCase inhibitors, the resistance raised after few years of application and is spreading rapidly to the wheat cropping area. Wheat monoculture system with highly reliance on chemical methods for weed control and several continuous years of application of herbicides with similar modes of action are recognized as the main reasons for resistance development in Iranian fields.

Zand et al. (2008) suggested that idosulfuron + metsulfuron, isoproturon + diflufenican, and pinoxaden (600 ml ha^{-1}) for the control of rigid ryegrass are available alternatives that could effectively delay the emergence of resistance. Applying appropriate crop rotation can maintain the efficiency of these herbicides for the control of rigid ryegrass in wheat farms.

There are some key points important for decision makers. The mechanism of herbicide resistance, the relative fitness of resistant and susceptible biotypes, the establishment pattern and competitive ability of the resistant plants, the longevity of pollen under variable conditions, the effective pollen dispersal distance, gene flow from resistant weeds to sensitive populations, genetic diversity and the population structure of weeds, and the distribution map of resistant biotypes are important subjects, and profound and expert knowledge of them would significantly improve the efficiency of resistance management programs. These issues, fortunately, are being investigated in research on herbicide weed resistance (Abdollahipour et al. 2013; Ghaseminejad et al. 2010; Kalami et al. 2014; Khoshayand et al. 2010; Najjari Kalantari et al. 2013; Tahmasebi et al. 2010).

With respect to the broad area of wheat crop production and continuous use of herbicides with the sole mechanism of action of ACCase inhibition, the provinces of West Azerbaijan, Tehran, Khorasan, Isfahan, Markazi, and Semnan are at risk of resistance development. In addition, because of continuous long-term use of tribenuron-methyl, resistance is also developing in broadleaf species. Evidence has recently shown the resistance of the turnipweed and wild mustard populations to this herbicide (Derakhshan and Gherekhloo 2012; Hatami et al. 2014, 2016).

There are no further reports of resistance development to the other herbicide groups in Iran. However, experiences from Iran and other countries indicate that continuous use of herbicides with the same site of action would result in weed resistance even to herbicide groups with lower resistance risk. Regular monitoring of fields in doubtful areas and providing good education and training for technicians and farmers to practice integrated methods would help to prevent or delay the development of resistance to herbicides.

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