SECTION I - HERBICIDE-RESISTANT WEEDS

Herbicide resistance is defined as the innate ability of a species to survive and reproduce after treatment with a dose of herbicide that would normally be lethal. It is important to differentiate herbicide resistance from herbicide tolerance, defined as the ability of a plant to compensate for the damaging effects of the herbicide with no physiological mechanisms involved. Resistant plants may be resistant to one class of herbicides within a group or to several herbicide classes within one group. For example, a biotype of wild oats (*Avena fatua*) that is resistant to fenoxaprop (an ACCase inhibitor) may be resistant to several other ACCase inhibitors. This is known as cross-resistance. Multiple resistance is defined as a biotype that is resistant to several groups of herbicides with different biochemical targets, such as triazines and ALS inhibitors. To control weeds with multiple resistance, it is necessary to use herbicides that are not in either of these groups, or some other alternative control strategy.

In recent years, herbicide-resistant weeds have developed from academic curiosities into serious management problems. Herbicide resistance in at least one weed species has occurred in almost every county in the tri-state area. Resistance seems to evolve fastest in continuous monoculture cropping situations, and if weeds like kochia and Russian thistle are involved, it can rapidly spread to adjacent cropland and rangeland, since seeds and pollen are widely disseminated. Herbicide-resistant weeds will continue to pose significant challenges in overall weed management schemes for the foreseeable future.

WHERE DO RESISTANT WEEDS COME FROM?

Herbicide-resistant weed biotypes are thought to develop from only one or a few plants already present within a population, usually at a very low frequency (maybe one in several million). Weeds, like every other organism, have inherent genetic variability that allows a few scattered individuals to survive herbicide treatment. These resistant individuals are not usually noticed during the first few years a herbicide is used. By repeatedly using the same herbicide over time, the applicator removes all the susceptible weeds and selects for the resistant plants. Then, depending on the selection intensity and life history of the weed species (see below), the resistant weed population will continue to grow and expand. Weed scientists and company representatives say that most growers won't complain until about 25% of the weeds in a given field become resistant.

The four most important factors controlling the appearance of resistant weeds are:

1) **Selection Intensity.** This term refers to how effective the herbicide is at killing weeds and how often the weed population is exposed to the herbicide. If the herbicide is highly effective, applied often, has long soil residual activity, and is the only practice for controlling a particular weed, then the selection intensity for resistance is very high. Under these conditions, selection of resistant weeds can occur within a few years (e.g., ALS resistance in kochia, ACCase resistance in grasses). In contrast, if a herbicide is only marginally effective on a certain species, is only applied sporadically, and/or has no soil residual activity, then the selection of resistant weeds will be slower.

2) **Weed Biology.** Some weed species have high levels of genetic variability, meaning that a single species consists of many different varieties or biotypes. Generally, weeds like kochia that are cross-pollinated (pollen is spread from one plant to another by insects or wind) have more diversity than those that are self-pollinated like wild oats. Weeds with more genetic variability generally develop resistance to herbicides sooner, since the initial frequency of resistant individuals before spraying is probably higher.

3) **Herbicide Mode of Action.** Many herbicides have similar modes of action and kill weeds by targeting the same enzyme. For example, sulfonylurea (Ally and Harmony GT) and imidazolinone (Pursuit and Assert) herbicides target the same plant enzyme, called ALS. As a result, continuous use of one herbicide (i.e., Glean) led to the evolution of weed biotypes that are resistant to most or all herbicides that target ALS. Rotating between herbicides (such as Harmony Extra and Upbeet, or Raptor and Express) that target the same site of action does not slow down the selection for resistant weeds.

4) **Genetics of Resistance.** Some sites of action will be selected for resistance to a herbicide quicker than others based on the amount of genetic variability for the enzyme within the weed population. For example, there may be more genetic variability for ALS or ACCase within weed populations prior to any herbicide application. As a result, the initial frequency of resistant individuals was higher and weed populations developed resistance quickly (within 3 to 4 years). In contrast, the genetics for some enzymes (i.e., EPSP synthase) are highly conserved. As a result, the frequency of resistant individuals prior to herbicide application is lower and resistance (i.e., to glyphosate) does not occur for many years.

PREVENTING HERBICIDE-RESISTANT WEEDS

The most important way to prevent the appearance of herbicide-resistant weeds is to rotate: rotate herbicides, rotate crops, and rotate management strategies. It is especially critical to rotate herbicides with different sites of action (Table 4.1). By using herbicides that have different enzyme targets each year, the chances of selecting for resistant weeds become much lower. For herbicides with residual soil activity, it is important to remember that they are still imposing selection pressure for resistant weeds as long as they are active in the soil. If available, a herbicide with a different mode of action should be rotated, or if not possible, other means of weed control should be substituted in non-application years. Crop rotations are a very useful strategy for avoiding resistance, providing that the herbicides used in different crops target different sites of action.

Table 4.1 List of herbicides grouped by site of action and herbicide families (chemically related). Examples of resistant weeds within Montana, Utah and Wyoming are also presented.

Group and Mode of Action[1]	Chemical Family	Common Name	Trade names(s)[2]	Resistant weeds found in MT, UT and WY
(1) ACCase Inhibitors - *prevents formation of fatty acids*				
	aryloxyphenoxy propanoates	clodinafop	Discover, Horizon	Wild oat, Persian darnel, Italian ryegrass
		diclofop	Hoelon	
		fenoxaprop	Puma, Tiller, Acclaim, Cheyenne, Dakota	
		fluazifop	Fusilade DX	
		quizalofop	Assure II	
	cyclohex-anediones	clethodim	Prism, Select	
		sethoxydim	Poast	
		tralkoxydim	Achieve	
(2) ALS inhibitors - *blocks protein synthesis*				
	imidazolinones	imazamethabenz	Assert	Kochia, Russian thistle, Wild oat, Italian ryegrass
		imazamox	Raptor, Motive	
		imazapic	Plateau	
		imazapyr	Arsenal	
		imazaquin	Scepter	
		imazethapyr	Pursuit, Lightning	
	sulfonylamino-carbonyltriazol-inones	flucarbazone-sodium	Everest	
		MKH 6561	Olympus	
	sulfonylureas	chlorsulfuron	Glean, Telar, Finesse	
		ethametsulfuron	Muster	
		halosulfuron	Permit	
		metsulfuron	Ally, Escort, Canvas, Finesse, Cimarron	
		nicosulfuron	Accent, Accent Gold, Basis, Basis Gold, Celebrity Plus	
		primsulfuron	Beacon, Exceed	
		prosulfuron	Peak, Exceed	
		rimsulfuron	Matrix, Accent Gold, Basis Gold	
		sulfometuron	Oust	
		thifensulfuron	Harmony GT, Pinnacle, Basis, Canvas, Cheyenne, Harmony Extra, Reliance, others	
		triasulfuron	Amber, Rave	
		tribenuron	Express, Canvas, Cheyenne, Harmony Extra	
		triflusulfuron	UpBeet	
	triazolopyrimides	chloransulam	FirstRate	
		diclosulam	StrongArm	
		flumetsulam	Python (Broadstrike)	

Table 4.1 continued

Group and Mode of Action[1]	Chemical Family	Common Name	Trade names(s)[2]	Resistant weeds found in MT, UT and WY
(3) Mitosis inhibitors - *interferes with new plant growth*				
	dinitroanalines	benefin	Balan, Team	
		ethalfluralin	Sonalan	
		oryzalin	Surflan, Rout	
		pendimethalin	Prowl, Pendimax, Squadron, others	
		trifluralin	Treflan, others	
(4) Synthetic auxins - *growth regulators*				
	phenoxy acetic acids	2,4-D	2,4-D, Campaign, Crossbow, Curtail, Landmaster BW, Scorpion III, Shotgun, Starane Salvo, Tiller, Weedmaster, others	
		2,4-DB	Butyrac	Kochia
		MCPA	MCPA, others	
	benzoic acid	dicamba	Banvel, Clarity	
	pyridines	clopyralid	Stinger, Reclaim	
		fluroxypyr	Starane	
		picloram	Tordon 22K	
	quinolines	quinclorac	Paramount, Drive	
(5) Photosystem II inhibitors - *blocks food producing pathway*				
	triazines	atrazine	Aatrex, others	
		cyanazine	Bladex	
		simazine	Princep, Derby	
	triazones	hexazinone	Velpar	Kochia
		metribuzin	Lexone, Sencor	
	uracils	bromacil	Hyvar	
		terbacil	Sinbar	
(6) Photosystem II inhibitors (different binding behavior than groups 5 and 7 but same site) - *blocks food producing pathway*				
	benzothiadiazoles	bentazon	Basagran, Galaxy Storm, Laddok	
	nitriles	bromoxynil	Buctril, Moxy, Broclean, Bronate, others	
	phenyl-pyradazine	pyridate	Tough	
(7) Photosystem II inhibitors (different binding behavior than groups 5 and 6 but same site) - *blocks food producing pathway*				
	amide	propanil	Stampede	
	ureas	diuron	Diuron, Direx, Karmex	
		linuron	Lorox, Linex, Linuron	
		tebuthiuron	Spike	

Table 4.1 continued

Group and Mode of Action[1]	Chemical Family	Common Name	Trade names(s)[2]	Resistant weeds found in MT, UT and WY
(8) Lipid synthesis inhibitors, but not ACCase inhibitors				
	thiocarbamates	cycloate	Ro-Neet	Wild oat
		EPTC	Eptam,	
		EPTC + safener	Eradicane	
		triallate	Far-Go	
Unknown site of action				
	no family name	difenzoquat	Avenge	Wild oat
(9) EPSP synthase inhibitors - *blocks protein synthesis*				
	glyphosate	glyphosate-ipa	Roundup, Rodeo, Glyphomax, Backdraft, Bronco, Campaign, Extreme, FieldMaster, Landmaster BW, FallowMaster, ReadyMaster ATZ	
		sulfosate	Touchdown	
(10) Glutamine synthetase inhibitors - *ammonia assimilation inhibitor*				
	phosphorylated amino acid	glufosinate	Liberty, Finale, Rely	
(14) PPO inhibitors - ***cell membrane disruptor***				
	diphenylether	fomesafen	Flexstar, Reflex	
	N-phenylthalimides	flumiclorac	Resource, Stellar	
	triazolinones	carfentrazone	Aim, Affinity	
		sulfentrazone	Authority, Spartan, Canopy XL	
(15) Unknown site of action				
	chloroacetamides	acetochlor	Harness, Surpass, TopNotch	
		alachlor	Lasso	
		dimethenamid	Frontier	
		metalochor	Dual, Dual II, Magnum	
		propachlor	Ramrod	
(22) Photosystem I electron diverters - ***cell membrane disruptor***				
	bipyridiliums	diquat	Diquat	
		paraquat	Gramoxone Extra, Starfire	

[1]Herbicide classification according to primary site of action and as organized by the Weed Science Society of America (WSSA).
[2]The use of trade names is provided for readers' information and inclusion of a trade name does not imply endorsement nor does exclusion imply disapproval.
[3]kochia, *Kochia scoparia* L.; Persian darnel, *Lolium persicum* L.; Russian thistle, *Salsola kali* L.; wild oat, *Avena fatua* L.

CASE HISTORIES OF HERBICIDE RESISTANCE

Kochia resistance to ALS inhibiting herbicides (*Ally, Amber, Canvas, Express, Finesse, Glean, Harmony GT, Harmony Xtra, Peak, Pursuit, and Raptor*) was found in 1988 and is present throughout nearly all small grain production areas of Montana, Utah, and Wyoming. The proportion of kochia populations resistant to ALS herbicides is greater than 50% in many parts of the Golden Triangle, Yellowstone River Valley, and Northeast Montana. As a result, herbicides such as Banvel, Bronate, Starane, should be mixed with ALS inhibiting herbicides to effectively manage kochia.

Because of the widespread occurrence of Glean-resistant kochia, most producers now use two- and three-way tank mixes of herbicides (2,4-D, Banvel, and a sulfonylurea or imidazolinone herbicide) for small grain weed control. However, 2,4-D has never been particularly effective on kochia, leaving only Banvel (dicamba) to control this troublesome species. In 1995, Banvel-resistant kochia plants were verified in several areas of Montana and Nebraska. Dicamba resistance is quite different than resistance to ALS inhibitors so management and prevention is different. There is concern and evidence to suggest dicamba resistant kochia may be tolerant/resistant to Starane. The Banvel- and Starane-resistant kochia populations don't seem to be spreading as fast as the Glean-resistant kochia. The continued appearance and spread of these and other resistant weeds points out the critical need for effective resistance management strategies. Producers have recently added Spartan (sulfentrazone) to their weed management toolbox as a way to manage problematic weeds including kochia, Russian thistle, buckwheat, common lambsquarters and pigweed spp. One of the most important features of Spartan is that it has a completely different biochemical target than other herbicides used in small grains. Therefore, it has good potential to control resistant kochia. Producers should be aware that Spartan is a residual herbicide and therefore has potential for crop injury.

After 15 to 20 years of continuous use of the herbicide FarGo, resistant wild oats began to appear at several locations in Montana and Canada. Unfortunately, these plants are also cross-resistant to Avenge, an unrelated wild oat herbicide. Many affected producers switched to Assert as an alternative herbicide with a different mode of action, and this strategy was effective for 5 years or so. However, Assert-resistant wild oats have now been identified in the state. In addition, some populations are cross-resistant to FarGo, Avenge, and Hoelon. These findings clearly demonstrate the possibility that producers may soon be trying to manage wild oat plants that are resistant to all available wild oat herbicides.

MANAGEMENT STRATEGIES FOR AVOIDING AND MANAGING HERBICIDE-RESISTANT WEEDS

1) Use herbicides only when necessary.

2) Rotate herbicides by mode of action. Do not make more than two consecutive applications of herbicides with the same site of action in the same field unless other effective control practices are also included in the management system.

3) Apply herbicides in tank-mixed, prepackaged, or sequential mixture. Combine herbicides which have different modes of action. It is important, however, that each herbicide used in a mixture has significant activity against potentially resistant weeds for this strategy to be effective. It is important to remember that, in many cases, the resistant species that developed were not the primary target weeds in those fields.

4) Rotate crops, particularly those with different life cycles to break up weed life cycles. For example, a rotation that includes winter wheat, alfalfa, and summer crops such as spring wheat, barley, corn, or dry beans is effective. DO NOT USE herbicides with the same mode of action in these different crops against the same weed unless other effective control practices are also used.

5) Use mechanical weed control practices wherever possible and identify the weeds that are present. Respond quickly to changes in weed populations so that you can restrict the spread of weeds that may have been selected for resistance.

6) Make postharvest weed control part of your regular field practices. Many weeds can exist unnoticed under a crop canopy, but have enough time to set viable seed once the crop is removed. This is a good way to reduce the overall number of weed seeds in the soil.

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