MISSISSIPPI STATE UNIVERSITY EXTENSION

Preventing and Managing Herbicide-Resistant Weeds in Turfgrass

Herbicide-resistant weeds are among the most troublesome issues facing the turfgrass industry. The loss of effective and economically viable herbicides results in lower quality turf and increases weed management budgets. *Herbicide resistance* is the inherited ability of a plant population to survive and reproduce following treatment with a normally lethal dose of herbicide.

This is not a new issue. Simazine- and atrazine-resistant groundsel (*Senecio vulgaris*) was reported as early as 1970 (Ryan, 1970). Simazine-resistant annual bluegrass (*Poa annua*) has plagued golf courses, sports fields, industrial turf, and sod production in the Southeast for more than two decades. As of 2014, more than 420 unique cases of herbicide-resistant weeds have been reported globally (Heap, 2014). That equates to 232 species having evolved resistance to 22 of 25 known herbicide sites of action and to 152 different herbicides. **Table 1** lists herbicide-resistant turfgrass weeds of Mississippi as of 2014.

Preventing and managing herbicide resistance is crucial to preserving key chemistries that turfgrass managers use to provide a playable and aesthetically pleasing turf surface. It is important to understand some basic terminology associated with herbicide resistance.

Mode of action (MOA) — Herbicides are active at one or more target sites within plants. Target sites are often enzymes that play a critical role in plant metabolism. The term "site of action" is used interchangeably with MOA; however, the terms have somewhat different connotations. The MOA is *how* a herbicide kills a plant. For instance, atrazine inhibits photosystem II, subsequently leading to a buildup of oxidative free radicals and a decrease in photosynthesis. Site of action is *where* the herbicide binds in order to kill a plant. Atrazine inhibits photosystem II by binding to a specific site of action, the quinone-B binding niche on the D1 protein.

Table 2 lists common turfgrass MOAs and example trade names. The fundamental principle for managing herbicide resistance is this: **repeatedly relying upon a single MOA selects for populations that are resistant.** Herbicides do not cause a mutation; they merely select for populations that tolerate a dose of herbicide. Subsequently, those populations expand in number.

Preventing herbicide resistance requires rotating herbicide MOAs in order to avoid the expansion of resistant populations. *Classification systems* have been developed to help herbicide applicators alternate MOAs. The most common are those developed by the Weed Science Society of America (WSSA) and the Herbicide Resistance Action Committee (HRAC).

The *WSSA system assigns* each herbicide a number based on the MOA. The *HRAC system* assigns a letter based on an alphabetized list of herbicide MOAs. Inhibition of acetyl CoA carboxylase (ACCase) is assigned the HRAC grouping of A. HRAC further amends herbicide groupings with a subscript numbering system that indicates different binding behavior. In the case of photosystem II-inhibiting herbicides, subclasses C_1, C_2, and C_3 indicate different behavior with a key binding protein.

Resistance can be either innate or evolved. *Innate resistance* is the ability of a plant to survive and reproduce following a herbicide application from the very first exposure. Innate resistance is also known as tolerance. *Evolved resistance* is a change in a specific weed species that was once susceptible to a herbicide but is now no longer controlled.

Table 1. Herbicide-resistant turfgrass weeds of Mississippi reported by the International Survey of Herbicide-Resistant Weeds (Heap, 2014).*

Resistant weed	Mode of action	Active ingredient	Trade name	State
goosegrass	mitotic inhibitors	pendimethalin trifluralin	Pendulum Treflan	MS
goosegrass	EPSP synthase inhibitors	glyphosate	Round-Up	MS
Poa annua	photosystem II inhibitors	atrazine simazine	Aatrex Princep	MS

*There likely are instances of resistance not yet reported.

Table 2. Modes of action commonly used by turfgrass managers.

Mode of action	WSSA	HRAC	Active ingredient	Trade name
acetyl CoA carboxylase (ACCase) inhibitors	1	A	diclofop clethodim	Illoxan Envoy
acetolactate synthase (ALS or AHAS) inhibitors	2	B	bispyribac-sodium foramsulfuron	Velocity Revolver
photosystem (PS) II inhibitors	5	C_1	simazine	Princep
photosystem (PS) I inhibitors	22	D	diquat	Diquat
protoporphyrinogen oxidase (protox) inhibitors	14	E	oxadiazon sulfentrazone	Ronstar Dismiss
carotenoid biosynthesis inhibitors	28	F_2	mesotrione topramezone	Tenacity Pylex
enolpyruvyl shikimate-3-phosphate (EPSP) synthase inhibitors	9	G	glyphosate	Round-up
glutamine synthase inhibitors	10	H	glufosinate	Finale
mitotic inhibitors	3	K_1	prodiamine	Barricade
cellulose synthesis inhibitors	29	L	indaziflam	Specticle
fatty acid and lipid biosynthesis inhibitors	16	N	ethofumesate	Prograss
synthetic auxins	4	O	dicamba	Banvel

Both innate and evolved resistance can be due to either target-site or nontarget-site resistance. *Target-site resistance* is due to a change in the molecular structure of the intended biochemical target that prevents the herbicide from binding. In the case of resistance to photosystem II inhibitors (atrazine, simazine, diuron, and amicarbazone), a molecular change prevents the specific binding of herbicide to the quinone-B binding site of the D1 protein. Similar modes of target-site resistance are also known to cause resistance to acetolactate synthase inhibitors (bispyribac-sodium, foramsulfuron), acetyl-CoA carboxylase inhibitors (fluazifop, diclofop, fenoxaprop), and mitotic-inhibiting herbicides (prodiamine, pendimethalin, oryzalin).

Nontarget-site herbicide resistance is a change in the ability of the herbicide to be absorbed, translocate throughout the plant, or be metabolized by the plant. Nontarget-site resistance may develop due to structural and chemical changes in plant leaves, such as thicker wax on leaf surfaces. It may also be due to reduced movement of the herbicide throughout the plant. For example, glyphosate resistance in horseweed (*Conyza canadensis*) has occurred due to reduced translocation of the herbicide (Koger and Reddy, 2005).

Prevent and Manage Resistance

Herbicide resistance is real, but you can take steps to prevent resistance and control already-resistant populations. The WSSA and HRAC classification systems are tools for developing resistance management strategies, but they should not be relied upon solely. Principally, proper cultural management enhances turfgrass vigor and reduces the reliance upon chemical weed control. Mechanical weed removal and application of nonselective herbicides via spot spraying are also crucial elements of resistance prevention and management.

Rotate Modes of Action

Repeat applications of the same MOA will select for resistant plants within a population. The more frequently herbicides with the same MOA are used, the more quickly resistant weed populations will develop. Rotation from Brand A to Brand B does not slow resistance development if both herbicides have the same MOA. Not only do managers have to rotate different herbicides, but they also have to use different MOAs. For instance, using atrazine in rotation with simazine is a futile approach, as both are photosystem II inhibitors (Group 5 herbicides). See **Table 3** for a more complete list of WSSA and HRAC classifications.

Use Tank Mixtures

If you use herbicide tank mixtures with different MOAs that are active on the same species, the weedy population would need to have tolerance to two different MOAs at the same time in order to survive. This may decrease the potential for resistance, but there are differences of opinion surrounding the issue. It is, however, likely that multiple MOAs improve the spectrum of weeds controlled, simultaneously reducing plants that need follow-up applications.

Table 3. Mode of action and classification of common turfgrass herbicides according to the Weed Science Society of America and the Herbicide Resistance Action Committee.

Timing	Mode of action	WSSA group	HRAC group	Common name	Trade name
pre	mitotic inhibition	3	K_1	dithiopyr	Dimension
pre	mitotic inhibition	3	K_1	pendimethalin	Pendulum
pre	mitotic inhibition	3	K_1	prodiamine	Barricade
pre	lipid biosynthesis inhibition	8	N	bensulide	Bensumec
pre	photosystem II inhibition	7	C_2	siduron	Tupersan
pre	protoporphyrinogen oxidase (PPO) inhibition	14	E	oxadiazon	Ronstar
pre/post	mitotic inhibition	15	K_3	dimethenamid	Tower
pre/post	mitotic inhibition	3	K_1	pronamide	Kerb
pre/post	mitotic inhibition	15	K_3	metolachlor	Pennant Magnum
pre/post	photosystem II inhibition	5	C_1	amicarbazone	Xonerate
pre/post	photosystem II inhibition	5	C_1	atrazine	Aatrex
pre/post	photosystem II inhibition	5	C_1	metribuzin	Sencor
pre/post	photosystem II inhibition	5	C_1	simazine	Princep
pre/post	cellulose synthesis inhibition	29	L	indaziflam	Specticle
pre/post	lipid biosynthesis inhibition	16	N	ethofumesate	Prograss
pre/post	carotenoid biosynthesis inhibition	28	F_2	mesotrione	Tenacity
pre/post	protoporphyrinogen oxidase (PPO) inhibition	14	E	flumioxazin	SureGuard
post	synthetic auxin	4	O	2,4-D	multiple
post	synthetic auxin	4	O	dicamba	Banvel
post	synthetic auxin inhibition of cell wall (cellulose) synthesis	4 27	O L	quinclorac	Drive
post	acetolactate synthase (ALS) inhibition	2	B	bispyribac-sodium	Velocity
post	acetolactate synthase (ALS) inhibition	2	B	foramsulfuron	Revolver
post	acetolactate synthase (ALS) inhibition	2	B	imazaquin	Image
post	acetolactate synthase (ALS) inhibition	2	B	metsulfuron	Manor
post	acetolactate synthase (ALS) inhibition	2	B	rimsulfuron	TranXit
post	acetolactate synthase (ALS) inhibition	2	B	sulfosulfuron	Certainty
post	acetolactate synthase (ALS) inhibition	2	B	trifloxysulfuron	Monument
post	acetyl CoA carboxylase (ACCase) inhibitors	1	A	diclofop	Illoxan
post	acetyl CoA carboxylase (ACCase) inhibitors	1	A	clethodim	Select
post	enolpyruvyl shikimate-3 phosphate (EPSP) synthase inhibition	9	G	glyphosate	Roundup
post	glutamine synthetase inhibition	10	H	glufosinate	Finale
post	photosystem II inhibition	6	C_3	bentazon	Basagran
post	photosystem I inhibition	22	D	diquat	Reward
post	photosystem I inhibition	22	D	paraquat	Gramoxone

Use Both Pre- and Post-Emergence Herbicides

Integrating both pre- and post-emergence herbicides into a weed management plan will diversify MOAs and eliminate weeds before they mature and develop seed. In such a plan, it would also be necessary to rotate both the pre- and post-emergence MOAs used each year. Atrazine followed by simazine would again be futile, because they have the same MOA. See Extension Publication 1532 Weed Control Guidelines for Mississippi.

Maximize Control and Minimize Escapes

It is important that herbicide applicators use the maximum labeled application rates in order to maximize control. Plants that escape control should be removed manually or chemically using a high-rate or nonselective spot spray application, according to label recommendations.

Optimize the Environment for Turf Plants

As always, the most important approach for weed management is to simply optimize the environment for the desired turf species. This will decrease the number of weeds that are actually treated by herbicides, thus decreasing the potential for resistance development.

Summary

Herbicide-resistant weeds are an increasing problem. An effective cultural and chemical management plan is required to achieve maximum weed control in turfgrass systems; however, emphasis should be placed on rotating herbicide modes of action and eliminating escaped weeds after herbicidal treatments have been applied. The Weed Science Society of America has developed a five-part training module on herbicide resistance awareness and education. Those modules can be accessed online at http://wssa.net/weed/resistance/.

References

Heap, I. 2014. The international survey of herbicide-resistant weeds. Available online at www.weedscience.org.

HRAC Website. 2014. Herbicide resistance action committee. Available online at www.hracglobal.com.

Koger, C.H. & K.N. Reddy. 2005. Role of absorption and translocation in the mechanism of glyphosate in horseweed (*Conyza canadensis*). *Weed Science, 53*: 84-89.

Ryan, G.F. 1970. Resistance of common groundsel to simazine and atrazine. *Weed Science, 18*: 614-616.

Weed Control Guidelines for Mississippi. 2014. Mississippi State Extension Service, Publication 1532.

Weed Science Society of America. 2014. Herbicide resistance training modules. Available online at http://wssa.net/weed/resistance/.

Publication 2844 (POD-06-22)

By **Jay McCurdy**, PhD, Associate Professor, Plant and Soil Sciences. Some ideas and concepts for this publication originate from an article that appeared in the November 2012 issue of *Golf Course Management*, written by Scott McElroy, PhD.

MISSISSIPPI STATE UNIVERSITY EXTENSION