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How to Manage Herbicide Resistance in Annual Bluegrass

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Figure 1. Annual bluegrass on a golf course.

Annual bluegrass (*Poa annua* L.) is one of the most problematic weeds of turfgrass lawns, athletic fields, and golf courses (Fig. 1). It is a winter annual that grows in clumps, can produce over 2,000 seeds per plant, and can persist at very low mowing heights. Annual bluegrass germinates in the fall and grows throughout the winter. During the spring, it produces an unsightly panicle type inflorescence (Fig. 2) that can disrupt play on putting greens.

Annual bluegrass grows in the winter when warm-season turfgrasses such as bermudagrass and zoysiagrass are dormant or semidormant. It does not have to compete with warm-season turfgrasses for light, water, and nutrients and thus it produces many seeds and can spread quickly if left untreated. There are many herbicides labeled for control of annual bluegrass, but because of restrictions due to overseeding, use site, turf species, and other reasons, turfgrass managers are often limited to just a few products, active ingredients, and modes of action (MOA).

Heavy reliance on herbicides to control annual bluegrass increases the likelihood that it will develop herbicide resistance. This is especially true if you routinely use only a single herbicidal mode of action.

Figure 2. Annual bluegrass clump and infloresence

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Tolerance versus resistance

Tolerance is a plant's *inherent* ability to survive and reproduce following a dose of herbicide that would be lethal to other plant species. For example, almost all types of annual bluegrass can survive an application of the herbicide 2,4-D, because annual bluegrass has a natural mechanism to withstand it. That is, annual bluegrass is naturally tolerant to this herbicide.

Resistance, on the other hand, is an *inherited* ability to survive a normally lethal dose of herbicide. For example, wild annual bluegrass biotypes will die following the application of an acetolactate synthase (ALS) inhibitor such as Revolver (foramsulfuron) or Monument (trifloxysulfuron-sodium). Annual bluegrass biotypes that survive these herbicides are considered resistant.

How do plants develop resistance to herbicides?

Many herbicides control weeds by inhibiting a single enzyme in susceptible plants. This enzyme is referred to as the *site of action*. Inhibiting this site of action disrupts a plant's essential processes and eventually kills it. A small change in this target site enzyme (i.e. target site mutation) can prevent the herbicide from inhibiting the enzyme. If the herbicide cannot inhibit the target enzyme, it cannot kill the plant and increasing the herbicide rate will usually not be effective. For example, in trials with a simazine-resistant biotype in Mississippi, it took 1,300 times more simazine to control the resistant than the nonresistant annual bluegrass. In addition to creating resistance to a particular herbicide, target site mutations usually enable plants to resist all other herbicides that inhibit the same target site (cross resistance), This is common for the ALS-inhibitor herbicides.

Resistance is not limited to changes (mutations) in target sites. Resistance can also occur when one weed's uptake, translocation, sequestration, or metabolic detoxification of a given herbicide is different from those of a susceptible biotype. Resistance that is non-target-site based can make plants resistant to multiple herbicide MOAs depending on the herbicide chemistries involved. This type of resistance has not been reported in turfgrass weeds; however, it is common agronomic crop weeds across the U.S.

Repeated use of herbicides with the same MOA makes resistance development more likely because weed populations have rare individuals that can survive them. You can eliminate these resistant individuals by varying the MOA you use. Conversely, if you use a single MOA the resistant weed will survive, multiply, and shift the weed population in favor of resistance. This is especially problematic in the case of a prolific seed producer such as annual bluegrass.

Herbicide resistance is widespread

Herbicide resistance in turfgrass weeds was first reported in Japan in 1982. The case involved an annual bluegrass that was identified as resistant to simazine—a photosystem-II (PSII) inhibitor. In a 2004 survey of 20 golf courses in Mississippi, 18 of the 20 locations reported simazine-resistant annual bluegrass. More recently, annual bluegrass biotypes with resistance to other PSII inhibitors (e.g. amicarbazone), microtubule inhibitors (e.g. prodiamine, pendimethalin, and dithiopyr), EPSP synthase inhibitors (glyphosate), and ALS inhibitors (e.g. foramsulfuron or trifloxysulfuron-sodium) have been reported in across the Southeast.

In most of these cases, annual bluegrass has only resisted one MOA. However, resistance to more than one herbicidal MOA (multiple resistance) is possible. The first annual bluegrass population with multiple resistance (to both ALS and PSII inhibitors) was recently reported in Tennessee. Multiple resistance severely limits options for annual bluegrass control. Weeds with resistance to multiple herbicide MOAs are increasing in croplands throughout the Midwestern U.S. This scenario could also occur in turfgrass cultures if we fail to address the problem of annual bluegrass resistance.

Scouting for herbicide resistant weeds

Identifying herbicide resistant weeds is an important management strategy. After you apply herbicides, look for weed control failures. Plants

Figure 3. Annual bluegrass plants that appear to be resistant to ALS-Inhibitors (Revolver, Monument, etc.) The plant showing injury is suscepticle while the two noninjured plants appear to be resistant.

displaying herbicide injury symptoms among unaffected plants is an indicator that plants may be herbicide resistant (Fig. 3). However, before assuming that weed survival (escapes) are due to resistance, remember that herbicide applications can also fail due to growth stage, application rate, uniformity, coverage, timing, temperature, etc.

If you suspect small populations of herbicide-resistant weeds, it is important to control them as soon as possible. This could involve removing them by hand or spot treating with a different herbicidal MOA. In theory, a population with target-site resistance originates from a single plant but goes undetected until the population is large enough to notice. For example, when a pre-emergence herbicide fails in patches that grow larger from year to year in the same place, the cause may be herbicide resistance. It is not feasible to spot treat every single weed that survives an application—a more practical approach is to look for clusters of annual bluegrass, especially among plants that were effectively controlled.

When you detect herbicide resistant weeds, it is important to use multiple MOAs in subsequent years because annual bluegrass seeds in the soil can be viable for at least six years. Again, once you identify annual bluegrass that is resistant to a certain MOA, you must vary the MOAs you use in subsequent years.

Preventing resistance

Using a single herbicidal MOA year after year makes it more probable that resistant populations will proliferate. Modern herbicides can control up to 100 percent of a given population, so resistant biotypes can flourish because all of the competition is eliminated. For a weed control program to prevent herbicide resistance, you should rotate MOAs. A survey of golf courses in Mississippi determined that 38 percent of courses that used simazine continuously for more than 5 years had resistant annual bluegrass populations. For courses with less than 5 years of continuous use, only 10 percent reported resistant populations.

Budgeting for weed control programs that use different MOAs from year to year can help reduce the development of resistant biotypes. Using herbicides with different MOAs separately or in tank-mixtures over the course of the same season is another good strategy. One way to deploy different MOAs in the same year is to apply pre-emergence herbicides such as prodiamine, pendimethalin, or dithiopyr, in combination with post-emergence herbicides. This strategy for preventing resistance is common in row crop systems, because pre- and post-emergence herbicides typically have a different MOA.

Herbicide options for annual bluegrass

There are 25 to 30 herbicides that are labeled for annual bluegrass, but there are only 10 different MOAs registered for use in turfgrass. Only a few of these are labeled for use on putting greens.

Pre-emergence and post-emergence herbicides labeled for annual bluegrass control are listed in Table 1 along with their trade names and MOA. To avoid developing resistant weeds or to manage existing ones, consider the MOA when selecting a herbicide. Post-emergence herbicides such as the ALS inhibitors, including Monument, (trifloxysulfuron-sodium) Revolver (foramsulfuron) and others, have been popular because they provide excellent selective control of annual bluegrass in established turf. If annual bluegrass develops resistance to an ALS inhibitor such as foramsulfuron,

rotating to trifloxysulfuron-sodium will not work because both herbicides have the same MOA. A product such as Tribute Total that contains three ALS-inhibiting herbicides (thiencarbazone-methyl, foramsulfuron, and halosulfuron-methyl) may not control ALS-resistant annual bluegrass either. In the case of annual bluegrass that is simazine-resistant (PSII-inhibitors), Xonerate (amicarbazone) may not provide control because it is also a PSII-inhibitor.

There are classification systems that easily reference the MOA and target sites for particular

Table 1. Herbicides registered for annual bluegrass control in warm-season turfgrass

Timing	Active ingredient	Trade name	Mode of action (HRAC Group, WSSA Group)
Pre	Bensulide[2,5]	Bensumec, others	Lipid synthesis inhibition (N,8)
	Benefin Dithiopyr[2,5] Oryzalin Pendimethalin Prodiamine Trifluralin+Benefin	Balan, others Dimension, others Surflan, others Pendulum, others Barricade, others Team	Microtubule assembly inhibition (K1,3)
	Flumioxazin[1, 4] Oxadiazon	SureGuard Ronstar, others	Protoporphyrinogen oxidase (PPO) inhibition (E,14)
	Dimethenamid Metolachlor	Tower Pennant Magnum	Very long chain fatty acid synthesis inhibition (K3,15)
Pre/Post	Indaziflam[1,3]	Specticle	Cellulose biosynthesis inhibition (L, 29)
	Pronamide	Kerb, others	Microtubule assembly inhibition (K1,3)
	Atrazine[1] Simazine[1]	Aatrex, others Princep	Photosystem-II inhibition (C1,5)
	Ethofumesate	Prograss	Lipid synthesis inhibition (N,8)
Post	Bispyribac-sodium Chlorsulfuron[1] Flazasulfuron[1] Foramsulfuron[1, 2] Imazapic Rimsulfuron[1, 2] Sulfosulfuron[3] Trifloxysulfuron[1]	Velocity Corsair Katana Revolver Plateau Rimsulfuron 25 DF Certainty Monument	Acetolactate synthase (ALS) inhibition (B,2)
	Amicarbazone Metribuzin[1]	Xonerate Sencor	Photosystem II inhibition (C1,5)
Post, non-selective	Glyphosate[4]	Roundup, others	Enolpyruvyl shikimate-3 phosphate (EPSP) synthase inhibition (G,9)
	Glufosinate[4]	Finale	Glutamine synthetase inhibition (H,10)
	Diquat[4]	Reward, others	Photosystem-I-electron diversion (D,22)

[1] Avoid tracking or movement after application onto adjacent sensitive grasses, including bentgrass putting greens
[2] Registered for use on ultradwarf bermudagrass putting greens
[3] Apply before annual bluegrass tillering for best results
[4] Apply to dormant bermudagrass only. See supplemental label for use in turfgrass.
[5] Some formulations labeled for use on bentgrass putting greens
Mention of trade name or commercial products in the publication is only for the purpose of illustration and does not imply recommendation or endorsement of any kind by the Texas A&M AgriLife Extension Service. Always read the label for up-to-date information on product use and turfgrass tolerance.

herbicides. The Weed Science Society of America (WSSA) and the Herbicide Resistance Action Committee (HRAC) have developed separate but similar systems. The WSSA classification is used more commonly in the United States and assigns herbicides to different numerical groups based on their sites of action. The HRAC system assigns a letter based on an alphabetized list of herbicide MOAs. The HRAC system further classifies herbicides with the same MOA using a numerical subscript that indicates different interactions with the enzyme target site.

Many agrochemical manufacturers display WSSA group numbers prominently on the product label (Fig. 4), while others may discuss the MOA on a different section of the herbicide label. With one exception (WSSA groups 5, 6, and 7 are all PSII-inhibitors that interact differently with the PSII proteins), herbicides with different WSSA group numbers have different MOA. This can simplify selecting herbicides with different MOAs.

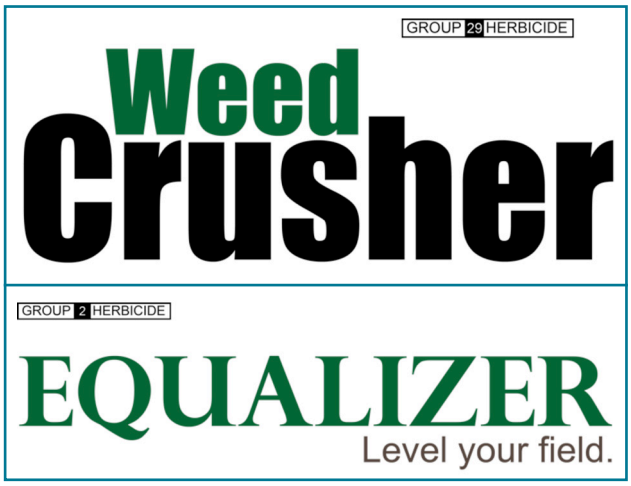

Figure 4. The examples above indicate how a WSSA group number is displayed on the label. This makes it easier to select herbicides with different modes of action. Group 29 indicates a product that inhibits the acetolactate synthase (ALS) enzyme. Group 2 indicates a product that inhibits cellulose biosynthesis. (These are not actual herbicide trade names and are provided for the purposes of demonstration)

Summary

Using a single herbicide MOA over many years makes it likely that herbicide-resistant weeds will develop. Tank mixing or making sequential applications of herbicides with different MOAs in the same season or rotating herbicide MOAs from one year to the next dramatically reduces the likelihood that herbicide-resistant annual bluegrass will develop.

When weeds are resistant to a certain MOA and the plan is to use only one herbicide for control, future applications should rely on a different MOA. If multiple herbicides will be applied in a given season, you should use at least two different MOAs.

Addressing current and potential herbicide resistance requires careful planning. If you suspect resistant plant populations, it is important to document when they emerge, where they occur, and what products have been used. Current herbicides are highly effective at very low application rates. However, given these product improvements, it is increasingly important to carefully select the MOA or combinations of MOAs that will best eliminate resistant biotypes and prevent new ones from developing.

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