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Herbicide-resistant weeds in turfgrass: current status and emerging threats

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Abstract

Herbicide-resistant weeds pose a severe threat to sustainable vegetation management in various production systems worldwide. The majority of the herbicide resistance cases reported thus far originate from agronomic production systems where herbicide use is intensive, especially in industrialized countries. Another notable sector with heavy reliance on herbicides for weed control is managed turfgrass systems, particularly golf courses and athletic fields. Intensive use of herbicides, coupled with a lack of tillage and other mechanical tools that are options in agronomic systems, increases the risk of herbicide-resistant weeds evolving in managed turfgrass systems. Among the notable weed species at high risk for evolving resistance under managed turf systems in the United States are annual bluegrass, goosegrass, and crabgrasses. The evolution and spread of multiple herbicide resistance, an emerging threat facing the turfgrass industry, should be addressed with the use of diversified management tools. Target-site resistance has been reported commonly as a mechanism of resistance for many herbicide groups, though non–target site resistance is an emerging concern. Despite the anecdotal evidence of the mounting weed resistance issues in managed turf systems, the lack of systematic and periodic surveys at regional and national scales means that confirmed reports are very limited and sparse. Furthermore, currently available information is widely scattered in the literature. This review provides a concise summary of the current status of herbicide-resistant weeds in managed turfgrass systems in the United States and highlights key emerging threats.

Introduction

Evolution of herbicide-resistant weeds is a major challenge to global agricultural production. Herbicide resistance was first reported in 1968 when common groundsel (*Senecio vulgaris* L.) was not controlled with the photosystem II (PSII)–inhibiting herbicides simazine and atrazine (Ryan 1970). Since then over-reliance on herbicides as the sole means of weed control has led to the rapid worldwide expansion of herbicide-resistant weeds. By the year 2000, over 300 unique cases of herbicide resistance—that is, species × site of action (SOA)—had been reported globally; this number grew to over 500 unique cases by 2019, the majority of which have been identified in major agronomic crops such as corn (*Zea mays* L.), soybean [*Glycine max* (L.) Merr.], and cereal crops (Heap 2019).

Although there were reports of common turfgrass weeds such as annual bluegrass and goosegrass evolving resistance to herbicides as early as the 1970s (Darmency and Gasquez 1983), these populations were not selected under typical turfgrass management practices, most importantly being part of perennial grass swards subjected to regular mowing. The first case of annual bluegrass resistance to PSII-inhibiting herbicides was identified along a sandy roadside in Normandy, France in 1975 (Darmency and Gasquez 1983). Similarly, goosegrass resistance to dinitroaniline herbicides was first identified in a cotton (*Gossypium* spp.) field in South Carolina (Mudge et al. 1984). The first report of a weed species evolving herbicide resistance under routine turfgrass management practices originated in annual bluegrass on golf courses in Mississippi; these populations survived treatment with the PSII-inhibiting herbicide simazine as a result of target-site mutation (Kelly et al. 1999). Since then, reports of herbicide resistance in turfgrass have become more common, with over 40 reports globally as of 2019 (Heap 2019). It is important to note that most of these turfgrass-specific reports have been weed populations found on golf courses, arguably the most intensively managed turfgrass system. Herbicide resistance on golf courses can severely compromise the aesthetic and functional quality of playing surfaces (Figure 1), whereas weed-infested athletic fields can be unsafe for athletes (Brosnan et al. 2014a). Failure to control weeds in sod production (due to herbicide resistance) can negatively affect the value of that industry valued at $1.6 billion (Haydu et al. 2006). The objective of this paper is to provide a review of past reports and offer

Figure 1. Glyphosate-resistant annual bluegrass (*Poa annua* L.) infesting a dormant bermudagrass (*Cynodon* spp.) golf course fairway in Rockford, TN.

thoughts on how continued evolution of herbicide-resistant weeds in turfgrass may affect weed management specialists on golf courses, sports fields, and lawns.

Acetyl CoA Carboxylase (ACCase)–Inhibitor Resistance in Turfgrass (WSSA Group 1)

Cases of ACCase inhibitor–resistant weeds in turfgrass systems are limited, and ACCase inhibitor–resistant annual bluegrass has yet to be confirmed. Derr (2002) identified a population of smooth crabgrass [*Digitaria ischaemum* (Schreb.) Schreb. ex Muhl.] on a golf course in New Jersey that was resistant to the ACCase-inhibiting herbicide fenoxaprop because of reduced sensitivity of the target enzyme (Kuk et al. 1999). McCullough et al. (2016c) identified a population of goosegrass in a field of centipede grass [*Eremochloa ophiuroides* (Munro) Hack] that evolved resistance to sethoxydim following 30 yr of POST application. This biotype was cross-resistant to clethodim, fenoxaprop, and fluazifop; however, it was susceptible to herbicides from other SOA groups including foramsulfuron (WSSA Group 2), glyphosate (WSSA Group 9), monosodium methanearsenate (MSMA, WSSA Group 0, and topramezone (WSSA Group 27). An Asp-2078-Gly substitution, previously associated with ACCase inhibitor resistance (Kaundun 2010), was identified in this resistant goosegrass biotype; moreover, the resistant biotype averaged two times greater metabolism of ^{14}C-diclofop in laboratory experiments. Other than quinclorac (WSSA Group 4), ACCase-inhibiting herbicides are the primary SOA used for POST grass control in warm- and cool-season turfgrass systems, making continued evolution of ACCase inhibitor resistance a concern for turfgrass managers.

Acetolactate Synthase (ALS)–Resistant Weeds in Turfgrass (WSSA Group 2)

Similar to other cropping systems, there are more instances of resistance to ALS-inhibiting herbicides in turfgrass than for any other SOA group (Heap 2019). Instances of ALS inhibitor resistance in annual bluegrass are the greatest in golf course populations, particularly in the transitional and southern zones of the United States and Australia (Brosnan et al. 2015, 2016; Cross et al. 2013; Heap 2019; McElroy et al. 2013). Mechanism of resistance in these populations has most often been target-site mutation, predominantly a Trp-574-Leu substitution on the ALS enzyme. However, Brosnan et al. (2016) identified annual bluegrass on a golf course with an Ala-205-Phe substitution that conferred cross-resistance to imidazolinone, sulfonylurea, triazolopyrimidines, sulfonylamino-carbonyl- triazolinones, and pyrimidinyl (thio) benzoate herbicides. All reported cases of ALS-inhibitor resistance in annual bluegrass originate from warm-season turfgrass systems for herbicides that typically provide nearly complete control of susceptible annual bluegrass biotypes. Cool-season turfgrasses are not tolerant to most of these herbicides, and there are no reported instances of ALS inhibitor resistance in annual bluegrass in cool-season turfgrass. Bispyribac-sodium is the only ALS-inhibiting herbicide commercialized for annual bluegrass control in cool-season turfgrass, but achieving effective annual bluegrass control without causing injury to desirable cool-season turfgrass with bispyribac-sodium is challenging, and use of this herbicide is not common (Lycan and Hart 2006; McCullough et al. 2009).

Several studies have demonstrated that alternative herbicide SOAs can be used to control ALS inhibitor–resistant annual bluegrass, particularly those applied PRE (Brosnan et al. 2015).

Among the selective POST herbicides, only amicarbazone (WSSA Group 5) and pronamide (WSSA Group 3) offer an alternative SOA for annual bluegrass control, and resistance to both amicarbazone and pronamide has been documented in this species (McCullough et al. 2017; Perry et al. 2012). Continued reliance on either of these herbicides for POST control of ALS inhibitor–resistant annual bluegrass will most likely select for biotypes with multiple resistance.

ALS-inhibitor resistance has been confirmed in other turfgrass weeds, including annual sedge (*Cyperus compressus* L.) and spotted spurge [*Chamaesyce maculate* (L.) Small] (McCullough et al. 2016a, 2016b). Evolution of ALS-inhibitor resistance in sedge species should be concerning to managers of both cool- and warm-season turfgrass, considering that other than sulfentrazone (WSSA Group 14), all other herbicides for selective POST control of sedge species in turfgrass are ALS inhibitors. In rice (*Oryza sativa* L.), ALS-inhibitor resistance evolved in yellow nutsedge (*Cyperus esculentus* L.) following continued use of halosulfuron (Tehranchian et al. 2015), one of the principal herbicides used for yellow nutsedge control in turfgrass.

Microtubule-Inhibitor (MTI) Resistance in Turfgrass (WSSA Group 3)

Annual bluegrass resistance to MTI herbicides is common throughout the transitional and southern zones of the United States (Breeden et al. 2017a; Brosnan et al. 2014b; Cutulle et al. 2009; Isgrigg et al. 2002; Lowe et al. 2001). Selection pressure for resistant biotypes has resulted from historical use of MTI herbicides during late summer and early autumn to control annual bluegrass PRE, and cross-resistance to MTI herbicides of various families has been documented (Cutulle et al. 2009). McCullough et al. (2017) reported an annual bluegrass biotype on a sod farm in Georgia that was resistant to pronamide, another MTI herbicide labeled for annual bluegrass control in warm-season turfgrass. This biotype survived POST applications of pronamide but was susceptible to PRE applications of pronamide at 0.56 and 1.28 kg ha^{-1}; the mechanism of resistance in this population was determined to be reduced absorption and translocation of foliar-applied pronamide. In addition to annual bluegrass, resistance to MTI herbicides has also been confirmed in goosegrass populations of turfgrass systems (Breeden et al. 2017b; McCullough et al. 2013).

Resistance to MTI herbicides is a recessive trait and therefore takes many years of selection to manifest in weed populations (Chen et al. 2019; Ghanizadeh et al. 2019). The slow evolution of resistance to prodiamine supports this notion; prodiamine was first registered for use in turfgrass in 1992, yet annual bluegrass populations evolving resistance to this herbicide were only documented after 10 yr (EPA 2019; Isgrigg et al. 2002). Additionally, aboveground growth of select MTI-resistant annual bluegrass biotypes is often reduced compared to susceptible biotypes (Brosnan et al. 2014b; Lowe et al. 2001), suggesting that there may be a fitness penalty associated with resistance to MTI herbicides.

Indaziflam is an alkylazine inhibitor of cellulose biosynthesis (WSSA Group 29) that has efficacy for controlling annual bluegrass and goosegrass with resistance to MTI herbicides in warm-season turfgrass (Brosnan et al. 2014b; McCullough et al. 2013). However, continued use of only indaziflam to control MTI-resistant annual bluegrass and goosegrass will select for populations with multiple resistance to both SOAs over time. Preventing MTI resistance is particularly important in the northern United States, considering that few labeled options are available for PRE control of annual grasses in cool-season turfgrass other than MTI herbicides. Further, enhanced soil degradation of MTI herbicides has been confirmed in Australia following consecutive applications without adequate rotation (Hole and Powles 1997). A similar pattern could be present for MTI herbicides such as prodiamine, pendimethalin, and dithiopyr that are regularly used in both warm- and cool-season turfgrass systems and warrants detailed investigation.

Synthetic Auxin Resistance in Turfgrass (WSSA Group 4)

Synthetic auxin herbicides are primarily used in turfgrass systems for controlling dicot weeds and are often applied in mixtures containing herbicides of various families within this SOA group. At present, synthetic auxin resistance in turfgrass is limited. Patton et al. (2018) identified a biotype of buckhorn plantain (*Plantago lanceolata* L.) in Indiana that evolved resistance to 2,4-D following continued use of 2,4-D + mecoprop + dicamba for multiple decades. Interestingly, this population was not cross-resistant to triclopyr, a pyridine carboxylic acid herbicide with an SOA similar to that of 2,4-D. Similarly, Russell et al. (2019) identified two buckhorn plantain biotypes resistant to 2,4-D in Pennsylvania that were not resistant to halauxifen-methyl, an arylpicolinate synthetic auxin. Resistance to triclopyr and clopyralid has been confirmed in lawn burweed (*Soliva sessilis* Ruiz & Pav.) populations in turfgrass systems in Australia (Heap 2019). Resistance was also confirmed for the synthetic auxin herbicide quinclorac in smooth crabgrass (Abdallah et al. 2006); reduced cyanide accumulation in foliar tissues was determined to be the mechanism of resistance in this biotype. Given the limited options for POST broadleaf weed control, particularly in cool-season turfgrass, continued evolution of auxin resistance in dicot weeds is concerning to turfgrass managers.

Photosystem II (PSII)–Inhibitor Resistance in Turfgrass (WSSA Group 5)

The first report of annual bluegrass resistance to PSII-inhibiting herbicides in turfgrass originated from a golf course in Japan in 1982 (Heap 2019). In the United States, Kelly et al. (1999) identified several PSII inhibitor–resistant annual bluegrass populations on golf courses in Mississippi that evolved from continued use of simazine in autumn for 12 yr consecutively; the mechanism of resistance in these populations was a Ser-264-Gly mutation on the D1 protein of the PSII reaction center that altered target-site binding of symmetrical triazine herbicides such as simazine. Perry et al. (2012) identified the same mutation conferring resistance to amicarbazone, a triazolinone herbicide labeled for POST annual bluegrass control. This response is most likely due to simazine and amicarbazone exhibiting similar binding kinetics on the D1 protein (Dayan et al. 2009), leading to cross-resistance. PSII-inhibitor resistance is concerning, as amicarbazone is the only PSII-inhibiting herbicide registered for use in cool-season turfgrass. However, similar to the ALS inhibitor bispyribac-sodium discussed above, amicarbazone is used sparingly by turfgrass managers because of limited efficacy against annual bluegrass at rates safe to cool-season turfgrass (McCullough et al. 2010). Mengistu et al. (2000) identified annual bluegrass biotypes in grass seed production fields that were resistant to the asymmetrical triazine herbicide metribuzin via a Val-219-Ile substitution on

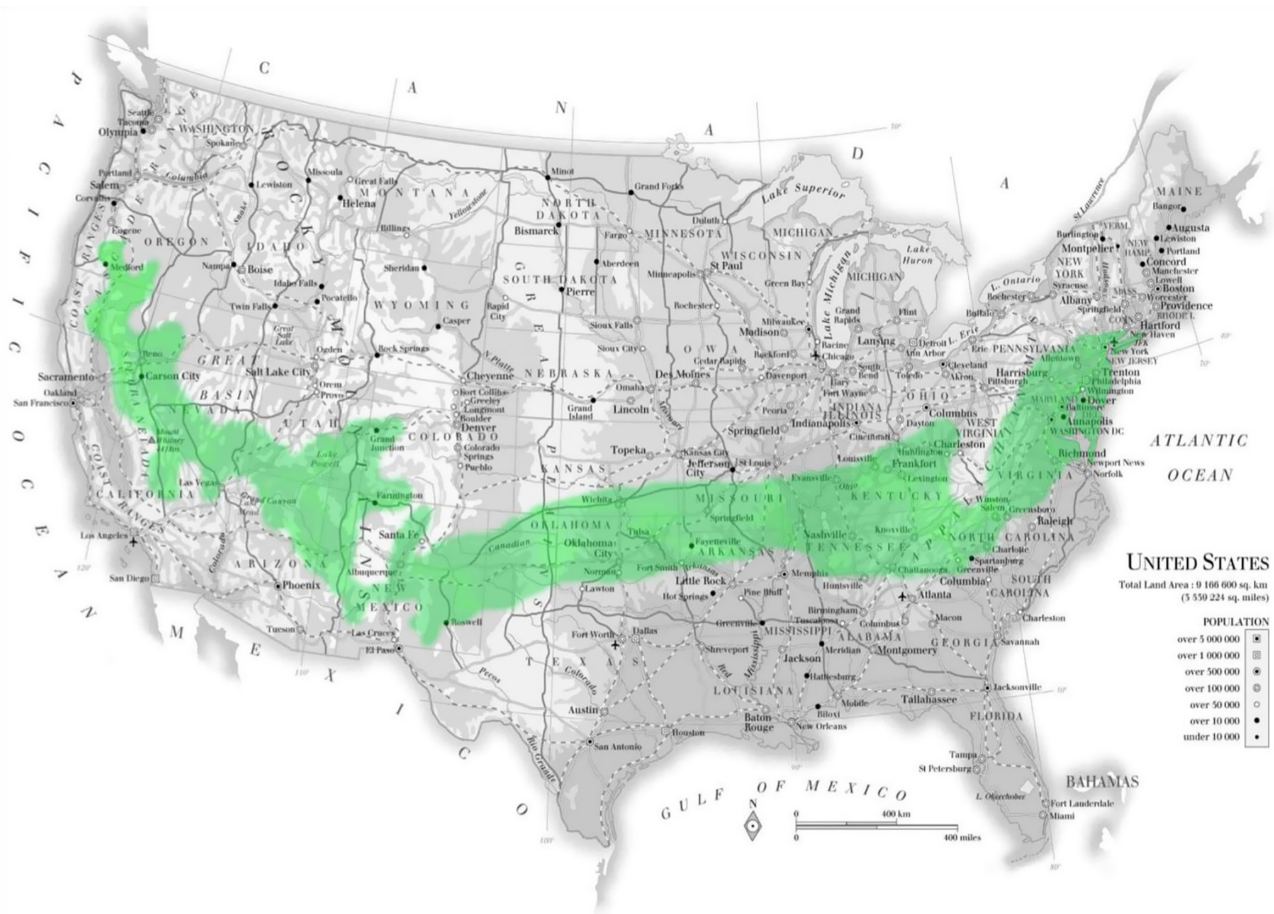

Figure 2. The transition zone region of the United States where glyphosate is applied to warm-season turfgrass species such as bermudagrass (*Cynodon* spp.) and zoysiagrass (*Zoysia* spp.) during winter dormancy. Image courtesy of Brandon Horvath, PhD.

the D1 protein; these biotypes were resistant to diuron as well. Although fitness experiments have not been conducted in PSII inhibitor–resistant annual bluegrass, mutations conferring resistance to this SOA have been generally associated with reductions in photosynthetic efficiency and overall growth potential (Kumata et al. 2001; Perry et al. 2012).

The extent of PSII inhibitor–resistant annual bluegrass in turfgrass systems is troubling. For example, Hutto et al. (2004) identified simazine-resistant annual bluegrass on 43% of golf courses in Mississippi. Simazine resistance was also identified in annual bluegrass on a sod farm in Tennessee (Brosnan et al. 2017b), which could perpetuate the issue over a wider geographical area. Moreover, annual bluegrass with multiple resistance to PSII inhibitors and other SOAs, particularly ALS inhibitors, has been reported (Brosnan et al. 2015, 2016). These multiple-resistant populations were selected via sole reliance on a single herbicide SOA for controlling PSII inhibitor–resistant populations, further highlighting the need for diversified management of annual bluegrass in turfgrass systems. PSII-inhibitor resistance has also been documented in goosegrass from continued use of metribuzin for POST control (Brosnan et al. 2008).

Lipid Synthesis–Inhibitor Resistance in Turfgrass (WSSA Group 15)

The benzofuran family herbicide, ethofumesate, provides POST annual bluegrass control in cool-season turfgrass systems (Dernoeden and Turner 1988). Over 20 annual bluegrass biotypes with resistance to ethofumesate have been reported in grass seed production fields in Oregon (Heap 2019; Mengistu et al. 2000). These populations were also resistant to triazine (WSSA Group 5) and urea (WSSA Group 5) herbicides. The propensity for annual bluegrass to develop resistance to ethofumesate is a matter of concern, as it is one of the few effective herbicide options available for selective POST annual bluegrass control in cool-season turfgrass. However, no ethofumesate-resistant biotypes have been reported in managed turfgrass swards.

Glyphosate-Resistant Weeds in Turfgrass (WSSA Group 9)

Selection pressure for glyphosate-resistant weeds in turfgrass systems is lower than in agronomic crops because of less frequent use of the herbicide. Broadcast applications of glyphosate over large acreages of maintained turfgrass typically occur only once annually in a limited geographical range, termed the transition zone (Figure 2), where warm-season turfgrass species such as bermudagrass (*Cynodon* spp.) and zoysiagrass (*Zoysia* spp.) grow during summer, but enter dormancy in winter when air temperatures fall steadily below 10 C (McCarty and Miller 2002). Winter dormancy allows desirable bermudagrass and zoysiagrass stands to tolerate treatment with a nonselective herbicide such as glyphosate. Moreover, competition from perennial turfgrass swards is thought to affect seed germination and seedling survival

of annual bluegrass, further minimizing the number of individuals exposed to glyphosate during these applications (Cross et al. 2015).

Nevertheless, several populations of annual bluegrass on golf courses in the US transition zone have evolved resistance to glyphosate (Binkholder et al. 2011; Breeden et al. 2017a; Brosnan et al. 2012; Cross et al. 2015). These populations have evolved under repeated use of glyphosate in dormant bermudagrass for several consecutive years without rotation of herbicides. Turfgrass managers have options for controlling these resistant populations, such as treatment with alternative nonselective herbicides labeled for POST annual bluegrass control during winter dormancy (e.g., glufosinate, diquat), or treating infested areas with herbicide mixtures varying in SOA during autumn while plants are still young (Breeden et al. 2017a). Mechanisms of resistance have not been studied widely in glyphosate-resistant annual bluegrass populations evolved on golf courses; however, differential shikimic acid accumulation has been reported when comparing several of these resistant populations to glyphosate-susceptible annual bluegrass (Breeden et al. 2017a; Brosnan et al. 2012; Cross et al. 2015). A Pro-106-Ala amino acid substitution on 5-enolpyruvylshikimate-3-phosphate synthase was reported in a glyphosate-resistant annual bluegrass biotype from South Carolina (Cross et al. 2015). More recently, Brunharo et al. (2019) identified EPSPS duplication as a mechanism of glyphosate resistance in annual bluegrass, albeit the biotype was not selected in a turfgrass system (instead originating in almond orchards).

Continued reliance on glyphosate for nonselective weed control during winter dormancy in turfgrass could also select for glyphosate resistance in other commonly occurring winter-annual weeds such as henbit (*Lamium amplexicaule* L.) and common chickweed (*Stellaria media* L.). There have also been numerous reports of goosegrass (a common summer-annual turfgrass weed) evolving resistance to glyphosate in various crop scenarios (Baerson et al. 2002; Kaundun et al. 2008; Lee and Ngim 2000; Mueller et al. 2011; Yu et al. 2015). One biotype of goosegrass from an oil palm (*Elaeis guineenis* Jacq.) nursery in Malaysia evolved resistance to three nonselective herbicides: glyphosate, glufosinate, and paraquat (Jalaludin et al. 2015). Considering that goosegrass is not present when broadcast applications of glyphosate are made during winter dormancy, selection pressure for glyphosate-resistant goosegrass biotypes in turfgrass will most likely continue to be low. However, an annual grassy weed evolving resistance to multiple nonselective modes of action is concerning. Occurrence of a similar phenomenon in annual bluegrass, which is regularly treated with nonselective herbicides during winter dormancy in the transition zone, would pose significant problems for turfgrass managers.

Protoporphyrinogen Oxidase (PPO)–Inhibitor Resistance in Turfgrass (WSSA Group 14)

Although resistance to PPO-inhibiting herbicides is not widespread in turfgrass systems, species that have evolved resistance are similar to those previously discussed for other modes of action. For example, Yu et al. (2018) identified a population of annual bluegrass on a golf course in Georgia that was resistant to POST applications of the PPO-inhibiting herbicide flumioxazin applied at a three- to five-tiller growth stage; however, this biotype was effectively controlled when flumioxazin was applied PRE. The mechanism of resistance in this PPO-resistant annual bluegrass biotype was associated with reduced lipid peroxidation and electrolyte leakage compared to herbicide-susceptible annual bluegrass. Similarly, McElroy et al. (2017) identified two biotypes of goosegrass resistant to PRE applications of the PPO inhibitor oxadiazon. However, both populations were completely controlled by PRE applications of indaziflam (WSSA Group 29) and prodiamine (WSSA Group 3). Despite the efficacy for controlling this PPO inhibitor–resistant goosegrass biotype, both indaziflam and prodiamine negatively affect root growth of warm-season turfgrasses, particularly in sandy soils (Jones et al. 2013). As a foliar-absorbed herbicide that has minimal effect on new root growth of desirable turfgrass species, oxadiazon therefore does not compromise establishment (Fagerness et al. 2002). Evolution of oxadiazon resistance is concerning, because, at present, turfgrass managers have few options for PRE control of summer-annual grasses that do not retard root growth of desirable turfgrass or introduce the potential for altered establishment, particularly following traffic stress on sports fields or winter injury on golf courses. Oxadiazon-resistant goosegrass has not been reported in cool-season turfgrass, but such cases would be extremely challenging to manage; prodiamine use is not common on golf courses and sports fields because of its potential to inhibit cool-season turfgrass root growth (Hummel et al. 1990), and indaziflam is not registered for use in cool-season turfgrass.

Multiple Herbicide–Resistant Weeds in Turfgrass

Multiple-herbicide resistance in weed populations is an emerging threat globally. Annual bluegrass and goosegrass are two important weed species infesting managed turfgrass systems in the United States that exhibit high propensity for evolving resistance to herbicides. Annual bluegrass ranks third globally in terms of the number of herbicide SOAs for which a weed species has evolved resistance, with resistance to nine SOAs documented to date (Heap 2019); goosegrass ranks fifth with resistance to eight SOA groups documented. In Tennessee, multiple resistance in golf course populations of annual bluegrass was reported for prodiamine and glyphosate (Breeden et al. 2017a), and for foramsulfuron/trifloxysulfuron and simazine (Brosnan et al. 2015). In Texas, annual bluegrass with three-way resistance to foramsulfuron/trifloxysulfuron, simazine, and pronamide was confirmed in a golf course population (Singh et al. 2017). Anecdotal reports suggest that multiple-herbicide resistance is more widespread than formally reported in annual bluegrass, especially in golf course populations, though systematic surveys are important to document the prevalence of such cases. In Australia, an annual bluegrass population was reported to exhibit five-way resistance to inhibitors of ALS, PSII, EPSPS, and microtubules, and an unknown SOA (WSSA Group 0) (Heap 2019); it is only a matter of time before such levels of multiple resistance occur in the United States if current herbicide use trends continue. In goosegrass, no multiple-herbicide resistance is reported as of now in the United States, but four-way resistance (ACCase inhibitor, PSI electron diverter, EPSPS inhibitor, and glutamine synthase inhibitor) was confirmed in an oil palm nursery in Malaysia (Heap 2019), highlighting the potential for multiple resistance evolving in this species. Other weed species infesting US turfgrass systems that show high risk for multiple resistance include crabgrasses (*Digitaria* spp.) and sedges (*Cyperus* spp.).

Non–Target Site Resistance (NTSR)

Weed resistance through NTSR mechanisms is a growing threat, because a broader resistance to even unrelated herbicide groups

is expected with these cases (Delye 2013). Although NTSR has been more commonly reported in weeds of row crop systems, documented cases of NTSR in turfgrass systems are limited as of now. Syvantek et al. (2016) identified a population of annual bluegrass in Alabama that was resistant to the PSII-inhibiting herbicides diuron, atrazine, and amicarbazone via NTSR. Laboratory experiments revealed that this biotype exhibited reduced absorption and translocation of atrazine, as well as enhanced atrazine metabolism, compared to both herbicide-susceptible biotypes as well as those resistant to PSII-inhibiting herbicides via target-site mutation. Brosnan et al. (2017a) observed that a biotype of annual bluegrass with target-site resistance to both ALS- and PSII-inhibiting herbicides (Brosnan et al. 2016) was less sensitive to methiozolin (WSSA Group 30) at doses ranging from 0 to 8,000 g ha^{-1} and postulated that this response may be due to NTSR. Genetic investigations of this population revealed a differential expression of genes encoding an ABC Type-2 transporter (sixfold), as well as several cytochrome P450 enzymes and catalases (Brosnan et al. 2019).

Evolution of herbicide resistance in weeds of both warm- and cool-season turfgrass is a growing concern for turfgrass managers. The evolution of cool-season weeds (such as annual bluegrass) in warm-season turfgrass and warm-season weeds (such as goosegrass) in cool-season turfgrass is most concerning. In these cases, target weeds are most competitive when the perennial turfgrass sward is least competitive, and thus turfgrass managers rely heavily on herbicides for weed control. Although herbicides with novel SOAs can aid in managing resistant weeds (Campe et al. 2018), few novel herbicides with new SOAs have appeared in recent years, and few are expected (Duke 2012). Turfgrass managers will need to implement diversified management tactics to preserve herbicides that are still effective for controlling weeds such as annual bluegrass and goosegrass. This could become challenging in a species such as annual bluegrass, where resistance via both target-site and non–target site mechanisms is increasing, similar to what has evolved within *Amaranthus* species of row crop systems.

Turfgrass weed control research should seek to better understand the value of nonchemical weed management in reducing herbicide resistance evolution. Cultural practices and improved turfgrass cultivars can increase turfgrass density and significantly reduce annual weed encroachment (Busey 2003). In theory, improved density of this perennial sward reduces the selection pressure herbicides impart, but we are not aware of research examining the effect of these strategies in delaying the onset of resistance or their economic value to the turfgrass manager. More research efforts are vital to develop truly integrated strategies for resistance management in turfgrass systems.

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