//WEED CONTROL

Herbicide resistant annual bluegrass

Coming soon to a course near you!

By Scott McElroy, Ph.D.

In my opinion, herbicide resistance is a major cause of reduced annual bluegrass control. Superintendents normally place the blame of unsuccessful annual bluegrass control on misapplication, mistiming and unfavorable environmental conditions — but in my research and observation, herbicide resistance is a widespread problem that is rarely considered as a possible cause of the problem.

In this article, I will define and explain herbicide resistance, relate this information to annual bluegrass and discuss how there are not easy solutions to solving the problem of herbicide resistance.

HERBICIDE RESISTANCE DEFINED

When a herbicide is labeled for use, there is a given expectation for weed control.

FIGURE 1

A classic case of a herbicide resistance pattern. In this case, a sulfonylurea herbicide was applied for postemergence control of *Poa annua*. Clearly some plants died as they should have and others did not. The pattern is seemingly random and there is no obvious spray pattern that could have caused the effect.

A labeled herbicide rate is established to provide an average level of control that is consistent based on potentially hundreds of research trials. Herbicide resistance arises when a weed species is able to survive and reproduce following a labeled herbicide treatment that has been confirmed to kill the given plant species. There can be varying degrees of resistance, from 1.5 to 2 times the normal labeled rate to resistance over 100 times the normal labeled rate. The degree of resistance often depends on the type of resistance pressure to which the plant has been exposed.

Herbicide resistance is a process of selection (some say natural selection, but herbicides are not very natural, so let's just say selection.) If one were to apply the same mode of action annually one would be applying selection pressure. Selection pressure with a herbicide eliminates the plants that are susceptible and only allows resistant plants to survive. Over several years one could eliminate a susceptible population entirely, only allowing for resistant plants to survive.

There are two basic ways in which a weed species can develop resistance — non-target site and target site. Target site resistance is a change in the protein or enzyme that a herbicide binds to or interferes with that causes plant death. Small changes of just one amino acid in a 500 amino acid enzyme can change the way a herbicide binds, thus preventing the herbicide from acting. Target site resistance is known to occur in mitotic-inhibiting herbicides (prodiamine, pendimethalin, oryzalin), PS II-inhibiting herbicides (atrazine, simazine, diuron, amicarbazone) and acetyl-CoA carboxylase inhibitors (fluazifop, diclofop, fenoxaprop.)

Non-target site herbicide resistance changes the way the herbicide behaves or is treated within the plant. Simply preventing the herbicide from absorbing in the plant would be a form of non-target site resistance. Other ways include changes that limit how the herbicide moves within the plant and the degradation of the herbicide within the plant. Such changes are actually not simple at all and would require multiple genetic changes to achieve such a resistance mechanism. Non-target resistance is most common in glyphosate resistant weeds.

Herbicide resistance is a process of selection... Selection pressure with a herbicide eliminates the plants that are susceptible and only allows resistant plants to survive.

HERBICIDE RESISTANT ANNUAL BLUEGRASS

Separate populations of annual bluegrass have developed resistance to almost all herbicides in use. The International Survey of Herbicide Resistance currently reports annual bluegrass resistance to photosystem II inhibitors (atrazine, simazine, diuron), photosystem I inhibitors (paraquat), inhibitors of very long-chain fatty acids (ethofumesate), mitotic-inhibiting herbicides (prodiamine, pendimethalin) and 5-enolpyruvate shikimate-3-phosphate inhibitors (glyphosate).

With this amount of resistance, there are very few herbicides that are still effective in all situations. Those

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What if annual bluegrass populations develop resistance to Velocity (bispyribac) which my research group has found? What are your options?

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herbicides that are effective are Specticle (indaziflam), Kerb (pronamide) and Finale (glufosinate.) See Ian Heap's website **http://www.weedscience.com** for more information on herbicide resistant weeds throughout the world.

WHY IS ANNUAL BLUEGRASS SO ADAPTABLE?

There are a few reasons why annual bluegrass is so adaptable to herbicide treatment. First, there is a lot of seed out there. Imagine all the potential millions of plants that are treated on one golf course in a given season. Now compound that with more golf courses and applying herbicides in successive years. There are an incalculable number of individual plants that would be treated. With that many plants, you eventually will find the one that is herbicide resistant (Figure 1.)

Second, annual bluegrass is a polyploid. Polyploids are species that are hybrids of two similar species or whose genomes have simply doubled. Think of it this way, humans (you and I, presumably) are diploids — this means that we have two sets of chromosomes. Polyploids have more than two sets of chromosomes.

In the case of annual bluegrass, it is a tetraploid — meaning that it has four sets of chromosomes — two sets from Poa infirma and two set from Poa supina, which are its ancestral parents.

But why is polyploidization beneficial? Think of it this way: If you have a diploid plant that has only two sets of the acetolactate synthase gene, one copy of the gene could be mutated to be resistant, which will eventually become two copies of the gene with the mutation if herbicide treatments continue to be applied.

The problem is that most mutations actually make the plant less fit or simply weaker compared to non-mutated plants. With a tetraploid, two copies of a gene from one parent can mutate and two copies of the same gene can remain in their fit form. So one plant, annual bluegrass in this case, can have the best of both worlds — it can make two fit copies of the gene for when no herbicide is being applied and makes two less fit herbicide resistant copies of the gene to help plants survive when herbicides are being applied.

WHAT IF...?

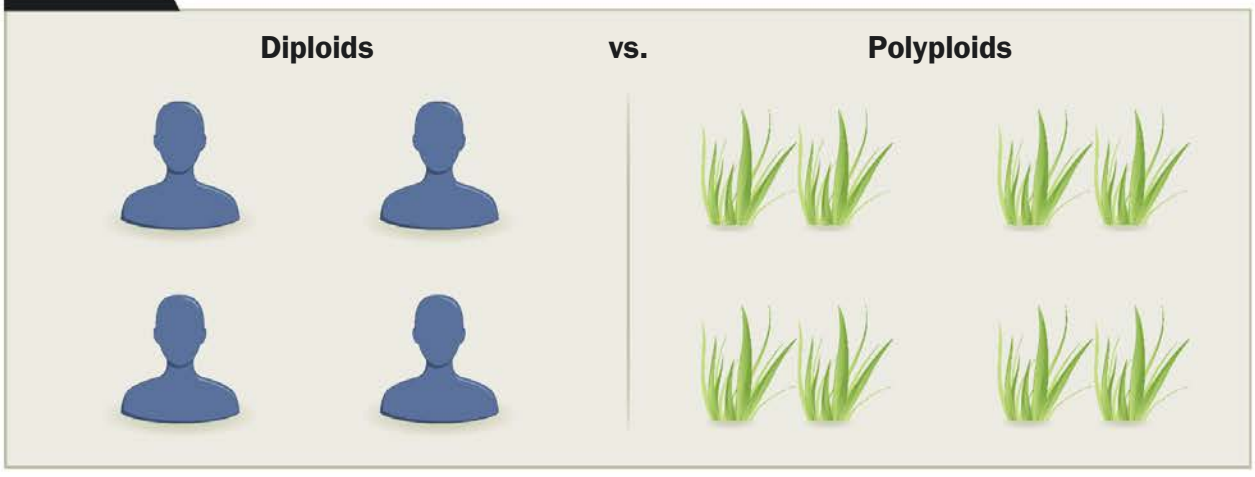
So what if one has an annual bluegrass population that develops resistance to a given herbicide or a class of herbicides with the same mode of action? The most immediate response is to change to another herbicide or herbicide mode of action, right?

Changing to another herbicide may not be that easy. Depending on the desirable turfgrass to be treated, adjacent turfgrass to the treated area and the level of weed control desired there maybe few to no options available.

Consider controlling annual bluegrass in bermudagrass surrounding a creeping bentgrass putting green. What if annual bluegrass develops resistance to dinitroaniline herbicides (prodiamine, pendimethalin) and is cross-resistant to dithiopyr? What are your options now?

One could use oxadiazon, but it has to be applied as a granular to dry turf to prevent injury and can only be applied as a liquid to dormant turfgrass. Glyphosate and glufosinate are options, but bermudagrass dormancy is questionable in greens surrounds and drift onto the putting green is possible. Sulfonylureas are options, but they are prone to off-target movement with surface water or tracking via tires and foot

FIGURE 3



Why is annual bluegrass so adaptable? One reason — it's a polyploid. Humans are diploids, which means we have two sets of chromosomes. Polyploids have more than two sets of chromosomes. Annual bluegrass actually has four sets of chromosomes.

traffic. Specticle (indaziflam) is a very effective preemergence herbicide for annual bluegrass, but it has off-target movement issues similar to sulfonylurea herbicides. Paclobutrazol can control annual bluegrass with multiple applications but bermudagrass green-up delay can occur. Sureguard (flumioxazin) is a new option that reportedly has less possibility for off-target movement, but lateral movement and traffic movement in turfgrass is difficult to predict. Xonerate (amicarbazone) is a new herbicide that controls annual bluegrass, but controlling larger plants may take two applications.

Confused yet? What would you choose to do?

Consider creeping bentgrass fairways or even greens. What if annual bluegrass populations develop resistance to Velocity (bispyribac), which my research group has found? What are your options?

One could use dinitroanilines or dithiopyr, but these herbicides present potential problems with root pruning and creeping bentgrass' ability to tolerate stressful conditions. Xonerate can be used in creeping bentgrass fairways, but repeat applications are needed in fairways, and very low rates and repeat applications are needed on greens. Even with these precautions, some injury is possible. Paclobutrazol can control annual bluegrass

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with repeat applications, but growth regulation and slight injury will occur. Oxadiazon, glufosinate, glyphosate, flumioxazin are not options.

The point is that changing to another herbicide or herbicide mode of action is not as easy as simply substituting another herbicide in for the one you lost. It is much more dynamic than that. In most situations one will have to totally restructure your application regime and modify your expectations for control. Trying to simply place a new herbicide in your current management plan is often the proverbial square peg in a round hole.

PREVENTING HERBICIDE RESISTANCE

When herbicide resistance prevention is discussed the first prevention strategy that is mentioned is "rotate modes of action." But what does this mean?

Let's use the example of using Specticle, which currently does not have any resistance issues, for preemergence control. Does rotating modes of action mean that in one year you should use Specticle and the next year use something completely different? And how often should you rotate modes of action — 1, 3, 5 years? Or do you change and treat half the acreage with Specticle and half with something else? What about tank-mixing another mode of action? Does that count as 'rotating herbicides?'

A final thought is that "spraying low herbicide rates increases resistance development." There is little to no evidence for this. It is possible that spraying low rates can aid in selection of non-target resistance mechanisms but not target site, but that is only speculative. One could also speculate that increasing herbicide rate, which increases selection pressure, could speed-up resistance development. In either case, one has to remember that herbicides do not cause the mutation, herbicides select for the mutation. Applying lower rates actually lowers the selection pressure.

Herbicide resistant annual bluegrass is a real and immediate problem in turfgrass management. Superintendents across the country are struggling with this issue and they do not even know it. Herbicide resistance will likely continue to develop in other weed species in the future as well.

Scott McElroy, Ph.D., is an associate professor of turfgrass science at Auburn University and can be reached at jsm0010@auburn.edu.

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