# HERBICIDE-RESISTANT WEEDS IN GEORGIA TURFGRASSES

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Selective weed control in turfgrasses essentially began with the discovery in the mid-1940's that 2,4-D would control dandelion in Kentucky bluegrass. In subsequent years, numerous herbicides have been registered for use in turfgrasses. The use of herbicides, in combination with timely cultural management practices, has significantly contributed to the overall aesthetic quality of turfgrasses.

Soon after the advent of other pesticides, certain species of insects and plant pathogens developed resistance, i.e., a species previously controlled by a specific pesticide was no longer controlled. Pesticide-resistance is not a new phenomenon. In 1908, resistance of San Jose scale to lime sulfur was observed. By 1957, entomologists had reported that 76 insect species were resistant to certain insecticides. A 1980 survey showed that 428 species of insects and related arthropods exhibited resistance to commonly used insecticides (1).

Now we are beginning to hear that some turfgrass weeds have developed resistance to herbicides. Actually, herbicide resistance is not new and was first reported in 1970, with the discovery that simazine, a triazine herbicide, no longer controlled groundsel (*Senecio vulgaris*), a previously susceptible species, in ornamental nurseries in Washington (3). As of 1989, 53 weed species were known to be resistant to various herbicide families (1). In 2003, 163 weed species are reported to be resistant to the various herbicide families. Within this group of herbicide-resistant weeds are several species that are major problem weeds in turfgrasses (Table 1).

Herbicide-resistance has been slower to develop, or to manifest itself, than insecticide- and fungicide-resistance. Possible reasons include: a) weeds normally complete only one life cycle per year, b) weeds are not as mobile as insects and disease pathogens, c) crop rotations that utilize different herbicide families and mechanical cultivation are routinely practiced in most crops, and, d) certain resistant weeds are less ecologically fit than their susceptible biotypes.

A common misconception is that continued use of the same herbicide causes a mutation to occur that enables the weed to become resistant to the herbicide. However, herbicides do not cause mutations. Research has shown that resistant individuals are naturally present at extremely low frequencies in a given population of a weed species. Continued use of the same herbicide over a period of years controls the susceptible biotypes, but allows the population of resistant biotypes to increase. The selection pressure exerted by the herbicide is analogous to a plant breeder selecting biotypes that are resistant (or more commonly tolerant) to various types of imposed selection stresses (drought, mowing height, diseases, insects, etc.). The end result of continued herbicide use for several consecutive years is a herbicide-resistant population of weeds. However, this statement is true only if resistant individuals are naturally present on the

site.

**A Classic Case of Herbicide-Resistant Weed Development in Turfgrass in Georgia.** In the mid-1980's goosegrass (*Eleusine indica*) resistance to the dinitroaniline herbicide family (trifluralin, pendimethalin, oryzalin, benefin, others) was reported in South Carolina (2). Annual use of dinitroanilines in cotton for 8 to 10 consecutive years was a major factor contributing to the development of this case of resistance. Prior to 1985, benefin was the only dinitroaniline herbicide registered for use on turfgrasses. However, in 1985 oryzalin, pendimethalin and trifluralin were registered for this use. Prodiamine, also a member of the dinitroaniline herbicide family was labeled for use in turfgrasses in the early 1990's. At about this same time, dithiopyr, a member of the pyridine herbicide family, was also registered for annual weed control in turfgrasses.

In 1992, a golf course superintendent in middle Georgia indicated that various dinitroaniline herbicides were not controlling goosegrass in bermudagrass fairways. Herbicide records were available only back to 1985, but did reveal that this golf course had used dinitroaniline herbicides alone or in combination with other herbicides for a period of seven consecutive years.

In 1993 and 1994, experiments were conducted on a common bermudagrass fairway at this golf course to determine if dinitroaniline-resistant goosegrass was present. Oxadiazon (Ronstar 2G), pendimethalin (several trade names), prodiamine (Barricade 65 WDG), oryzalin (Surflan 4AS) and dithiopyr (Dimension 1EC) were applied at maximum labeled rates to separate plots either a single or sequential application.

A single or sequential application of oryzalin, prodiamine and pendimethalin at the maximum labeled rate did not control goosegrass. Additionally, dithiopyr did not control goosegrass either as a single or sequential application. Pendimethalin, prodiamine and oryzalin are members of the dinitroaniline herbicide family and have the same basic mode-of-action, e.g. inhibition of a specific phase of cell division. Dithiopyr belongs to the pyridine herbicide family but has a mode of action similar to dinitroaniline herbicides. Because of their similar modes-of-action, rotating to dithiopyr was not an acceptable control strategy for dinitroaniline-resistant goosegrass.

This research showed that either a single and sequential application of oxadiazon provided $\geq$ 90% goosegrass control on this site. Oxadiazon belongs to the oxadiazole herbicide family and has a mode-of-action totally different than the dinitroaniline herbicides and dithiopyr (Table 1). Therefore, on sites where dinitroaniline- or dithiopyr-resistant goosegrass is present, rotation to oxadiazon, or other herbicides that have a different mode-of-action than the dinitroanilines and dithiopyr will be necessary to effectively control this biotype of goosegrass.

Additional research conducted at this site showed that diclofop (Illoxan) and MSMA + metribuzin (Sencor Turf) effectively controlled dinitroaniline- and dithiopyr-resistant goosegrass (data not shown). This group of herbicides also has a different modes-of-action than the dinitroanilines and dithiopyr.

Similar to that observed where resistance occurred in cotton fields, continued annual use of

dinitroaniline herbicides for a period of several years contributed to the development of resistant goosegrass. Therefore, herbicide resistant weeds can occur in turfgrasses and turfgrass managers should include a herbicide-resistant weed control strategy in their weed management plan.

We do have herbicide-resistant weeds in turfgrasses in Georgia. Most notable are: annual bluegrass resistance to triazines, ethofumesate, dinitroanilines and dithiopyr; and, goosegrass resistance to diclofop-methyl, dinitroanilines and dithiopyr. Once resistance occurs, the only practical option for control in turfgrasses is to rotate to a herbicide that has a different mode-of-action than the herbicide previously used (Table 2). Rotating to a different herbicide in the same chemical family is not effective, as members of the same family have the same mode-of-action. Additionally, increasing the rate of the herbicide is not an effective option as true herbicide resistance is absolute and is not related to tolerance. In the case of dinitroaniline-resistant goosegrass in Georgia, rotation to oxadiazon, diclofop or MSMA + metribuzin effectively controlled this weed. This group of herbicides have a different mode-of-action than dinitroaniline herbicides and dithiopyr.

For several years, dinitroaniline herbicides have been widely used by turfgrass managers to effectively control annual bluegrass, goosegrass, crabgrass (*Digitaria* spp.) and other annual weeds. While annual bluegrass and goosegrass resistance to this herbicide family and dithiopyr has been documented, it should be noted that there are no documented cases of crabgrass resistance to these herbicides. Also, herbicide resistance is not a widespread problem at this time, and, no one can accurately predict that resistant weeds will occur on every turfgrass site. If there are no resistant individuals in a given population of weed species, then the problem will not occur. However, rather than take chances, a basic principle of pest control, i.e. pesticide rotation, should be practiced. By following this basic principle, turfgrass managers can continue to depend upon the effective, low-cost control that the dinitroaniline and triazine herbicides have provided in the past.

Herbicide-resistant weeds are a real phenomena. Factors that contribute to the development of herbicide-resistant weeds include a) continued annual use of herbicides with similar modes-of-action, b) lack of use of herbicides with different modes-of-action, and c) allowing herbicide-resistant weeds to reseed. Most herbicide resistant weeds have not developed in a short period of time. In the case of dinitroaniline-resistant goosegrass in turfgrasses, dinitroaniline herbicides were annually used for a period of 8 to 10 years. Continued annual use of the same herbicide is one of the primary reasons why herbicide-resistant weeds are increasing in various crop systems, and have the potential to increase in turfgrasses. Other reasons include the development of herbicides that have a single site mode-of-action, and use of herbicides that provide several months of residual weed control activity.

Herbicide resistant weeds are not a major problem in turfgrasses. However, they can become a serious problem unless turfgrass managers begin to employ herbicide-resistant weed management strategies. Management practices that discourage, or help to prevent, herbicide-resistant weeds are: a) use of herbicides that have a different mode-of-action, b) use of tank-mixes or combinations of herbicides that have different modes-of-action, c) controlling weeds that escape preemergence herbicide treatments with postemergence herbicides that have a different mode-of-action, and d) preventing seed production by hand roguing (where practical).

The dinitroaniline and triazine herbicide families have provided and continue to provide economical annual grass control in established turfgrasses. However, we have documented that herbicide-resistant weeds can occur in turfgrasses. There is a natural tendency is to continue to use pesticides that have been successful in the past. Insecticide and fungicide rotation is routinely practiced on golf courses and other turfgrass sites. If turfgrass managers are to prevent herbicide-resistance from becoming widespread, then herbicide rotation will also need to be routinely used as well.

## References


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2. Mudge, L. C., B. J. Gossett and T. R. Murphy. 1984. Resistance of goosegrass (Eleusine indica) to dinitroaniline herbicides. Weed Science 32:591-594.

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


**Table 1. Examples of herbicide-resistant weeds.**

| Herbicide-Resistant Weed | Herbicide | Brand Name(s) |
|---|---|---|
| Goosegrass | diclofop-methyl,<br>fluazifop,<br>dithiopyr,<br>pendimethalin,<br>prodiamine,<br>oryzalin,<br>benefin,<br>trifluralin | Illoxan<br>Fusilade II<br>Dimension<br>Pendulum, others<br>Barricade<br>Surflan<br>Balan<br>Team (also contains benefin) |
| Annual bluegrass | dithiopyr,<br>pendimethalin,<br>prodiamine,<br>oryzalin,<br>benefin,<br>trifluralin,<br>ethofumesate,<br>simazine,<br>atrazine,<br>metribuzin | Dimension<br>Pendulum, others<br>Barricade<br>Surflan<br>Balan<br>Team (also contains benefin)<br>Prograss<br>Princep, others<br>Aatrex, others<br>Sencor |
| Italian ryegrass | diclofop-methyl,<br>sulfometuron,<br>glyphosate | Illoxan<br>Oust<br>Roundup, others |
| Horseweed (marestail) | glyphosate | Roundup, others |

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|---|---|---|
| Smooth crabgrass | quinclorac | Drive |

**Table 2. Mode-of-action of commonly used turfgrass herbicides.**

| Herbicide Family | Common Name | Brand Name[1] | Application Timing | Mode-of-Action |
|---|---|---|---|---|
| Aryloxyphenoxy propionate | Diclofop, Fluazifop-P, Quizalofop-P | Illoxan, Fusilade II, Assure II | Postemergence | Inhibition of fatty acid synthesis. |
| Bipyridilium | Diquat | Reward | Postemergence | Cell membrane disruption through formation of hydroxyl and lipid radicals |
| Dinitroaniline | benefin, oryzalin, pendimethalin, prodiamine, trifluralin | Balan, Surflan, Pendulum, others, Barricade, Treflan | Preemergence | Inhibits cell division by binding to tubulin which prevents polymerization of microtubules at the growing end of the tubule. |
| Imidazolinone | Imazaquin | Image | Postemergence | Inhibits the enzyme, acetolactase synthase, a key enzyme in the synthesis of the branched chain amino acids isoleucine, leucine and valine. |
| Organic Arsenical | MSMA, DSMA | Bueno 6, others, DSMA 4, others | Postemergence | Not well understood. Known to uncouple energy transfer during the production of ATP. |
| Oxadiazole | oxadiazon | Ronstar | Preemergence | Inhibits Protox enzyme which leads to formation of lipid radicals which disrupts cell membrane integrity |
| Pyridine | dithiopyr | Dimension | Preemergence, Postemergence | Inhibits cell division in the late prometaphase stage by binding to a microtubule associated protein. Does not bind to tubulin. |
| Sulfonylurea | chlorsulfuron, sulfometuron, metsulfuron foramsulfuron trifloxysulfuron | Corsair, Oust, Manor, Blade, Revolver, Monument | Preemergence, Postemergence | Inhibits the enzyme, acetolactase synthase, a key enzyme in the synthesis of the branched chain amino acids isoleucine, leucine and valine. |
| Triazine | atrazine, metribuzin, simazine | Aatrex, Others, Sencor 75 Turf, Princep, Others | Preemergence, Postemergence | Inhibits electron transport during the light-dependent phase of photosynthesis. Membrane disruption ensues due to formation of toxic lipid radicals. |

[1] Brand names are used only for information. Other products may be available with the same active ingredient.