Herbicide-Resistant Weeds in Turfgrasses

by Tim R. Murphy

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. Subsequently, 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, some species of insects and plant pathogens developed pesticide resistance, i.e., a pesticide that had previously controlled a species would no longer do so. This was not a new phenomenon. Resistance of San Jose scale to lime sulfur had been observed in 1908. 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).

Herbicide resistance was first reported in 1970, with the discovery in ornamental nurseries in Washington that simazine, a triazine herbicide, no longer controlled groundsel (*Senecio vulgaris*), a previously susceptible species (3). As of 1989, 53 weed species were considered to be resistant to various herbicide families (1).

Herbicide-resistance has been slower to develop, or to manifest itself, than insecticide- and fungicide-resistance. Some possible reasons for this 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 with most crops, and, d) certain resistant weeds are less ecologically fit than their herbicide-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 individuals of resistant biotypes are naturally present at extremely low frequencies in most populations 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 use of one herbicide 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.

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. Other herbicide families or herbicides in which resistance has been noted for other weed species include the triazines (atrazine, simazine, others), organic arsenicals (MSMA, DSMA), sulfonylureas (chlorsulfuron, sulfometuron, others), imidazolinones (imazaquin, others), diclofop, quizalofop, fluazifop and paraquat. Many of these herbicides are registered for use in various turfgrass species.

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 available back to 1985 revealed that dinitroaniline herbicides had been used on this golf course alone or in combination (with other herbicides) for seven consecutive years.

Experiments were conducted on a common bermudagrass fairway at this golf course in 1993 and 1994 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 the maximum labeled rates to separate plots in either single or successive applications. In both 1993 and 1994, the initial herbicide application was in mid-February, and a second application was made approximately eight weeks later in mid-April. Goosegrass control was assessed at four and five months after the initial February application in 1993 and 1994, respectively. A control rating of less than 80% is not considered to be commercially acceptable.

A single application of oryzalin, prodiamine or pendimethalin at the maximum labeled rate did not control goosegrass (see Table 1). Subsequent applications of these herbicides at these same rates also did not provide control. The sequential application program is equivalent to a rate that is twice the labeled maximum. Additionally, dithiopyr did not control goosegrass as either a single or sequential application.

Pendimethalin, prodiamine, and oryzalin, being all members of the dinitroaniline herbicide family, have the same basic mode-of-action: inhibition of a specific phase of cell division (see Table 2). Dithiopyr, although belonging to the pyridine herbicide family, has a mode of action similar to the dinitroaniline herbicides. Because of their similar modes-of-action, rotating to dithiopyr proved not to be an effective strategy for controlling dinitroaniline-resistant goosegrass.

Single and sequential applications of oxadiazon provided 90% or better goosegrass control in 1993 and 1994 see (Table 1). Oxadiazon belongs to the oxadiazole herbicide family and has a mode-of-action totally different from the dinitroaniline herbicides and dithiopyr. Therefore, on sites where a dinitroaniline- or dithiopyr-resistant biotype of goosegrass is present, rotation to oxadiazon, or other herbicides that have a mode-of-action different from the dinitroanilines and dithiopyr should provide effective control.

Additional research conducted at this site showed that diclofop (Illoxan®) and MSMA + metribuzin (Sencor Turf®) effectively controlled dinitroaniline- and dithiopyr-resistant goosegrass. Herbicides in this group also have different modes-of-action from those of the dinitroanilines and dithiopyr.

Similar to what was observed in the development of pesticide resistance in cotton fields, exclusive use of dinitroaniline herbicides for a period of several years allowed the population of resistant goosegrass biotypes to flourish. Therefore, turfgrass managers should include a herbicide-resistant weed-avoidant control strategy in their weed management plan.

Table 1. Goosegrass control with selected preemergence herbicides.

Goosegrass Control[2]			
Herbicide	Rate[1] lbs. ai/acre	1993 %	1994 %
Oryzalin	3.0	0	30
Oryzalin	3.0 + 3.0	0	10
Prodiamine	0.75	27	53
Prodiamine	0.75 + 0.75	40	37
Pendimethalin	3.0	20	47
Pendimethalin	3.0 + 3.0	13	47
Dithiopyr	0.5	7	27
Dithiopyr	0.5 + 0.5	0	13
Oxadiazon	4.0	93	96
Oxadiazon	4.0 + 4.0	94	100
No Herbicide	--	0	0
LSD (0.05)	--	29	42

[1] Herbicides applied in single or sequential applications. Single applications were made in mid-February. Sequential applications were made in mid-February and mid-April.

[2] Control ratings were recorded four months after application in 1993 and five months after application in 1994.

Table 2. Mode-of commonly used herbicides.

Herbicide Family	Common Name	Brand Name[1]	Application Timing	Mode-of-Action
Aryloxyphenoxy propionate	Dielofop, Fluazifor-P, Quizalofop-P	Illoxan®, Fusilade II®, Assure II®	Postemergence	Inhibition of fatty acid synthesis.
Bipyridilium	Paraquat	Gramoxone Extra®	Postemergence	Cell membrane disruption through the formation of hydroxyl and lipid radicals.
Dinitroaniline	benefin, oryzalin, pendimethalin, prodiamine, trifluralin	Balan®, Surflan®, Pre-M®, others Barricade®, Treflan®	Preemergence	Inhibits cell division by binding to tubulin which prevents poly-merization 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.
Pyridine	dithiopyr	Dimension®	Preemergence Postemergence	Inhibits cell division in the late prometaphase stage by binding to a microtubule associated pro-tein. Does not bind to tubulin.
Sulfonylurea	chlorsulfuron, sulfometuron	Glean®, Tolar®, Oust®	Preeemergence, 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	Astrex®, others, Sencor 75 Turf® Princep®, others	Preemergence, Postmergence	Inhibits electron transport during the light-dependent phase of photosynthesis. Membrane dis-ruption 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.

Once resistance occurs, the only effective option for control in turfgrasses is to rotate to a herbicide that has a different mode-of-action than the herbicide previously used. Rotating to a different herbicide in the same chemical family is not effective, as members of the same family usually 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 discussed above, rotation to oxadiazon, diclofop or MSMA + metribuzin effectively controlled this weed. This group of herbicides has a different mode-of-action than dinitroaniline herbicides and dithiopyr.

Dinitroaniline herbicides have been used widely for several years by turfgrass managers to control goosegrass, crabgrass (*Digitaria* spp.) and other annual weeds. While goosegrass resistance to this herbicide family and dithiopyr has been documented, there are no documented cases of crabgrass resistance to these herbicides. And goosegrass resistance has not become a widespread problem at this time.

No one can accurately predict whether resistant goosegrass will occur on every turfgrass site. If there are no resistant individuals in a given population of goosegrass, 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 dinitroaniline herbicides have provided in the past.

Herbicide-resistant weeds are, nonetheless, a real phenomenon. Factors that contribute to their emergence include a) consistent 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 weed populations take a long time to develop. In the case of dinitroaniline-resistant goosegrass in turfgrasses and cotton, dinitroaniline herbicides were used exclusively for periods of eight to ten years. Continued annual use of the same herbicide is one of the primary reasons why herbicide-resistant weeds are appearing more frequently in various crop systems, and why they have the potential to increase in turfgrasses. Other reasons include the development of herbicides that have a single-site/ narrow-spectrum mode-of-action, and use of herbicides that provide several months of residual weed control activity.

Herbicide resistant weeds have not been a major problem in turfgrasses. They can proliferate, however, unless turfgrass managers begin to employ herbicide-resistant weed management strategies. Specific management practices that discourage, or help to prevent, the development of herbicide-resistant weed populations are: a) sequential use of herbicides with differing modes-of-action (rotation), 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) (where practical) preventing seed production by hand roguing.[3]

This examination of goosegrass resistance to the dinitroaniline herbicides and dithiopyr is not meant to suggest that it is time to push the "panic button." Nor does it indicate that these herbicides are no longer effective. The dinitroaniline herbicide family has provided and should continue to provide economical annual grass control in established turfgrasses. Their efficacy notwithstanding, herbicide-resistant weeds can become a problem in turfgrasses. There is a natural inclination to continue to use pesticides that have proved themselves effective in the past. Insecticide and fungicide rotation is now routine practice on turfgrass sites. If turfgrass managers are to hold herbicide-resistance in check, then herbicide rotation will have to become routine practice as well.

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

[3] For additional insights on increasing the effectiveness of weed-control management practices see: Lambert B. McCarty, "Winter Weed Control in Southern Turf - Early Detection, Recognition and Action are Key," *TurfGrass TRENDS*, 4-11 (NOV 95), pp. 9-13.