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Herbicide Resistance: Development, Prevention, and Recognition

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Herbicides are applied either to the foliage of growing weeds (postemergence) or to the soil to prevent germination (preemergence). Postemergent foliar herbicides are either contact or systemic chemicals.

Contact herbicides weaken and disorganize the plant cell membranes, causing leakage and eventual localized death. An example of a contact herbicide is Gramoxone. Systemic herbicides include the phenoxy herbicides (for example, 2,4-D) and dicamba and glyphosate. Systemic herbicides are translocated throughout the plant to their sites of physiological action.

Preemergent herbicides *(e.g.,* Casoron, Surflan, Ronstar) are applied either to the soil or growing-medium surface and are usually absorbed by root systems or by emerging shoot tips as they make their way through the soil surface during seed germination. Preemergents must be dissolved in the soil/medium solution in order to be effective.

Herbicide resistance is the genetic capacity of a weed population to survive an herbicide treatment that under normal use would effectively control that weed population (Martin *et al.,* 2001). Herbicide-resistant plants are present in a population in very small numbers. The repeated use of one herbicide allows these few plants to survive and reproduce (Mallory-Smith *et al.,* 1999).

Another way herbicide resistance may develop that is thought to be less contributory is a genetic mutation. This occurs after the herbicide has been applied and provides resistance to the herbicide (Hager *et al.,* 1998). A dilemma that is facing many producers of various agricultural and horticultural crops, in many states, is the development of biotypes of weed species that are resistant to herbicides.

A biotype is defined as a population of weeds within a given species that possesses certain traits not common to the entire population. Although the problem of herbicide-resistant weeds is not widespread in nursery culture, nursery managers should become knowledgeable about how resistance develops so that the prevalence of resistant weeds can be minimized (Hager *et al.,* 1998).

Weed resistance to herbicides is not unique (Kendig and Fishel, 1996). Insecticide-resistant insects, fungicide-resistant fungal pathogens, and antibiotic-resistant bacteria were discovered long before herbicide-resistant weeds. Spreading dayflower *(Commelina diffusa)* was the first herbicide-resistant weed identified. It was found to be resistant to 2,4-D in 1957, in a Hawaii sugar field (Mallory-Smith *et al.,* 1999).

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Herbicide resistance was first reported in nurseries in 1970 (Prather *et al.,* 2000). Common groundsel *(Senecio vulgaris)* in a Washington state tree nursery was shown to be resistant to herbicides in the triazine chemical class (Prather *et al.,* 2000).

Not every weed control failure you encounter in the future will be due to resistant weeds. However, the appearance of herbicide resistance in plants today is increasing at an exponential rate (Prather *et al.,* 2000) and is affecting the nursery operator's choice of herbicide. Besides triazine resistance, there are biotypes of 172 weed species expressing resistance to 16 other herbicide classes (Prather *et al.,* 2000). Worldwide, there are more than 249 herbicide-resistant weedy biotypes in 47 countries (Martin *et al.,* 2001).

Mode-of-Action

Herbicides have a specific target site, a place in the plant where herbicides bind and inhibit function (Hall *et al.,* 1999). Herbicides with the same mode-of-action will have the same translocation (movement) pattern, produce similar injury symptoms (Ross and Childs, 1996), and frequently have the same application method, constraints, and even toxicological profile (Hall *et al.,* 1999).

One or more vital processes in the plant must be disrupted in order for herbicides to kill a weed. Many herbicide target sites are enzymes; however, there are exceptions. Some vital metabolic plant processes that herbicides act on include photosynthesis (capture of light and carbohydrate synthesis), amino acid and protein synthesis, fat (lipid) synthesis, pigment synthesis, nucleic acid synthesis (RNA - DNA essential to information storage and transfer), and maintenance of membrane integrity (Ross and Childs 1996). Disruption of mitosis (cell division) in plant meristems (shoots or roots), disruption of meiosis (division resulting in gamete and seed formation), interference of uptake of ions and molecules, translocation of ions and molecules and transpiration are some other ways herbicides act to kill plants. In North America, herbicides are divided into groups based on target site. Herbicides with different modes-of-action should be rotated.

Some commonly used ornamental herbicides can be divided into 13 different groups based on mode-of-action, as indicated in Table 1:

- Lipid synthesis inhibitors at acetyl CoA carboxylase (ACCase), such as the cyclohexanediones and aryloxyphenoxy-propionates.
- Branched-chain amino acid synthesis inhibitors at acetolactate synthase (ALS), such as the imidazolines and sulfonylureas.
- Microtubule assembly inhibitors or seedling root inhibitors, such as the dinitroanalines (DNAs) and pyridines.
- Plant growth regulators that mimic auxins in the plant, such as the

phenoxy acetic acids, benzoic acids, and picolinic acids.

- Photosystem II inhibitors such as the triazines.
- Photosystem II inhibitors with different binding behavior, such as the benzothiadiazoles.
- Photosystem II inhibitors with yet another different binding behavior, such as the ureas.
- Aromatic amino acid inhibitors at EPSP synthase such as glycines.
- Glutamine synthesis inhibitors such as phosphinic acid.
- Photosystem I electron diverters such as bipyridyliums.
- Cell division seedling shoot inhibitors such as chloroacetamides and acetamides.
- Cell wall synthesis inhibitors such as benzamides and nitriles.
- Cell membrane disruptors such as diphenyl ethers, oxadiazoles, and N-phenylphthalimides.

How Does Resistance Develop?

Some management practices increase the likelihood of developing herbicide resistance. Resistance is more likely to occur when the same herbicide, or herbicides having the same modes of action, are used repeatedly.

It was thought that not using herbicides from the same family would prevent resistance. However, this is no longer the case (Mallory-Smith *et al.,* 1999). For example, two chemically different groups of herbicides, the sulfonylureas and imidazolinones, have the same site of action.

Using these two herbicide families repeatedly could lead to herbicide resistant biotypes, even though different families of herbicides are being used. This type of resistance development would be called cross-resistance. Table 1 indicates chemical families that have been shown to result in cross-resistance among weed species (Prather *et al.,* 2000).

The two herbicides indicated earlier are applied at different times. The imidazolinones are preemergents, and the sulfonylureas are postemergents. Many nursery growers think applying herbicides at different times, *i.e.,* pre- or post-emergence, means they are applying herbicides with different modes-of-action; however, the example listed previously indicates that this is not true.

The current recommendation is to rotate herbicides with different sites of action. Do not make more than two consecutive applications of herbicides with the same site of action to the same field **(http:// agguide.agronomy.psu.edu/sect1/sec13f.htm, 2002).**

Two consecutive applications can mean single applications for two years *(i.e.,* spring 2003 and spring 2004), or two split applications in one year *(i.e.,* spring 2003 and fall 2003).

Monocultures often encourage the use of the same herbicide and are more likely to develop herbicide-resistant weeds (Martin *et al.,* 2001). Resistance is most likely to develop in annual weed species since they produce high numbers of seeds (for example, pigweed, lambsquarters, and foxtail). Resistance often occurs in the

Text continues on page 95

Table 1. Some Herbicides Registered for Use in Outdoor Ornamentals and Non-Crop Areas.

Mode of action, herbicide family, trade and common name, and application timing are indicated. PSI and PSII indicate photosystem I and II. Chemical families marked with the same herbicide resistance activity code (HRAC) have been shown to result in cross-resistance among weed species. (Source for codes, Prather *et al.*, 2000).

Mode-of-Action and HRAC	Herbicide Family	Trade Name	Active Ingredient Common Name	Application Timing
Amino acid synthesis Inhibitor (ALS inhibitor)				
A	Imidazolinones	Plateau	Ammonium salt	Pre
		Image	Ammonium salt	Pre
A	Sulfonylurea	Manage	Halosulfuron-methyl	Post
Amino acid synthesis inhibitor (EPSP synthase inhibitor)				
B	Glycine	Rattler	Glyphosphate isopropylamine salt	Post
		Roundup Pro DRY	Glyphosate monoammonium salt	Post
		Roundup Pro	Glyphosphate isopropylamine salt	Post
		Mirage	Glyphosphate isopropylamine salt	Post
		Silhouette	Glyphosphate isopropylamine salt	Post
		Prosecutor	Glyphosphate isopropylamine salt	Post
		Touchdown Pro	Sulfosate	Post
Cell wall synthesis inhibitor				
C	Benzamide	Gallery 75 DF	Isoxaben	Pre
		Snapshot 2.5 TG	Isoxaben + Trifluralin + Fertilizer	Pre
C	Nitrile	Casoron 4G	Dichlobenil	
Cell membrane disruptor (inhibits PS I)				
D	Bipyridylium	Reward L&A	Diquat dibromide	Post
		Starfire	Paraquat dichloride	Post
		Boa	Paraquat dichloride	Post
		Gramoxone	Paraquat dichloride	Post
Cell membrane disruptor (Inhibit PPO enzyme)				
E	Diphenyl ether	Goal 2XL	Oxyfluorfen	Pre
		OH II	Oxyfluorfen + Pendimethalin	Pre
		Rout 3G	Oryzalin + Oxyfluorfen	Pre
		Regal 0-0	Oxadiazon + Oxyfluorfen	Pre

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Mode-of-Action and HRAC	**Herbicide Family**	**Trade Name**	**Active Ingredient Common Name**	**Application Timing**
E	Oxadiazole	Ronstar G	Oxadiazon	Pre
		Regal Star	Oxadiazon + Prodamine	Pre
		Kansel Plus	Oxadiazon + Pendamethalin	Pre
Cell membrane disruptor				
F	N-phenyl-phthalimides	SureGuard	Flumioxazin	Pre
		Broadstar*	Flumioxazin	Pre
Fatty acid syntheses Inhibitor (ACCase inhibitors)				
G	Cyclohexanediones	Vantage	Sethoxydim	Post
		Envoy	Clethodium	Post
G	Aryloxyphenoxy-propionate	Fusilade II	Fluazifop-p-butyl	Post
		Acclaim Extra	Fenoxaprop	Post
Growth regulator				
H	Phenoxy	Salvo	2,4-D Isoctyl (2-ethylhexyl) ester	Post
		Weedar 64	2,4-D Amine	Post
H	Benzoic acid	Banvel	Dicamba	Post
H	Picolinic acid	Garlon	Triclopyr	Post
		Lontrel T&O	Clopyralid	Post
		Stinger 3L	Clopyralid	Post
Glutamine synthesis inhibitor				
I	Phosphinic Acid	Finale	Glufosinate ammonium	Post
Non-mobile Photosynthetic Inhibitors (Inhibits PSII)				
J	Benzothiadiazole	Basagran	Sodium bentazon	Post
		Basagran T/O	Sodium bentazon	Post
Mobile PS II inhibitor				
K	Triazine	Atrazine 4L	Atrazine	Pre
		Atrazine 90Df	Atrazine	Pre
		Simazine 90WDG	Simazine	Pre
		Simazine 4L	Simazine	Pre
		Simazine 90 DF	Simazine	Pre
		Princep 4L	Simazine	Pre
		Caliber 90	Simazine	Pre

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Mode-of-Action and HRAC	**Herbicide Family**	**Trade Name**	**Active Ingredient Common Name**	**Application Timing**
Mobile PS II inhibitor (Different binding behavior than triazines)				
L	Urea	Diuron 4L	Diuron	Pre
		Diuron 80DF	Diuron	Pre
		Diuron 80WDG	Diuron	Pre
Mitotic disruptors; Microtubule assembly inhibitors (Root meristem inhibitor)				
M	Pyridine	Dimension	Dithiopyr	Post
		Dimension EC	Dithiopyr	Post
		Dimension Ultra 2SC	Dithiopyr	Post
		Dimension Ultra WSP	Dithiopyr	Post
M	Dinitroaniline (DNAs)	Barricade 65WG	Prodiamine	Pre
		Barricade 4FL	Prodiamine	Pre
		Kerb 50WP	Prodiamine	Pre
		Hurdle 3.8 ASC	Pendimethalin	Pre
		AquaCap	Pendimethalin	Pre
		Pendulum 2G,	Pendimethalin	Pre
		Pendulum 3.3 EC	Pendimethalin	Pre
		Pre-M 60DG	Pendimethalin	Pre
		Corral 2.68G	Pendimethalin	Pre
		Rout	Oryzalin + Oxyfluorofen	Pre
		Snapshot 2.5 TG	Isoxaben + Trifluralin + Fertilizer	Pre
		Team Pro	Benefin + Trifluralin + Fertilizer	Pre
		Surflan AS T/O	Oryzalin	Pre
		Surflan Coated Granules	Oryzalin	Pre
		XL 2G	Oryzalin + Benefin	Pre
		Oryzalin 4 Pro	Oryzalin	Pre
		Treflan 5G	Trifluralin	Pre
		Treflan HFP	Trifluralin	Pre
		Trifluralin EC	Trifluralin	Pre
		OH II	Oxyfluorfen + Pendimethalin	Pre

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Mode-of-Action and HRAC	**Herbicide Family**	**Trade Name**	**Active Ingredient Common Name**	**Application Timing**
Cell division inhibitor (Seedling shoot inhibitor)				
N	Acetamide	Pre-Pair	Napropamide	Pre
		Devrinol 2G	Napropamide	Pre
		Devrinol 50DF	Napropamide	Pre
N	Chloroacetamide	Pennant Magnum 7.62 EC	Metolachlor	Pre
N	Benzamide	Kerb WSP	Pronamide	Pre

Note: * Indicates product not currently labeled.

absence of other control practices such as cultivation, other physical controls, and/or cultural controls (Hager *et al.,* 1998).

An integrated weed-management approach is important for all weed-control management programs but is especially relevant to managing herbicide resistance. Resistance also occurs often with herbicides that have the greatest efficacy on a specific species (Martin *et al.,* 2001).

The intense selection pressure imposed by these efficacious herbicides results in the survival of only the resistant individuals. These then are the only individuals that pass their genes to the next generation (Martin *et al.,* 2001).

Using herbicides that do not persist in the soil for a long time and are not applied repeatedly in a growing season reduces the selection of herbicide resistant weeds (Mallory-Smith *et al.,* 1999). Many nursery growers are concerned about herbicide residues, of soil-persistent herbicides, affecting the subsequent crop.

Many herbicides are broken down in soils by microbes. Some herbicides decompose within three to four weeks, like 2,4-D. Other compounds are more resistant to microbial breakdown and can persist longer than one year, like diuron and atrazine.

In general, any condition that favors the growth of microbes will hasten herbicide decomposition. Temperature, moisture, aeration, organic matter content, pH, minerals present, concentration of the herbicide, species of microbes present, the herbicide used, and soil preconditioning are all important factors.

A warm, moist, fertile soil is generally ideal for microbial breakdown of herbicides. These conditions are not always experienced. In a drought situation, where no irrigation is supplied, the herbicide remains on the dry surface of the soil. Microbes are not active in these conditions, and the herbicide not only fails to provide weed control but also may persist until the next season and cause crop damage.

Prevention of Herbicide Resistance

One way to prevent resistance is to not use persistent preemergents and to test to determine if herbicide residues exist on a site. One persistent ornamental preemergent is Casoron, which is tied up by organic matter and slowly decomposed by soil microbes.

Casoron residue damage would generally show up as one-directional rooting. Other injury symptoms of Casoron include leaf yellowing or veinal, interveinal, marginal, or overall chlorosis.

Simazine and Princep are other persistent preemergents that can buildup in soils with repeated applications over several years.

Simazine injury appears as yellowing or veinal, interveinal, marginal, or overall chlorosis. Injury appears first in the new growth, since the chemical is translocated to the growing point. The whole leaf may become chlorotic at high concentrations. Atrazine residues are more serious in northern climates as inactivation is very slow below 75 F°.

Soils can be chemically analyzed for herbicide residues, but this is expensive, complicated, and can be done only in specialized laboratories. Moreover, the results of the analysis do not indicate the effects on the next crop.

An inexpensive and fairly reliable way to determine herbicide carryover or residue is to make a crop biological assay — a bioassay. A bioassay determines the biological activity of a substance by testing its effect on a test plant or organism.

Bioassays should also be done if you are renting a parcel of land for which you do not know the cropping history.

Soil sampling for a crop bioassay is similar to sampling for fertilizer levels. Samples should be gathered from several areas of the field. Remember that the assay is only as reliable as the sample collected. If possible, a nontreated or check soil sample should be taken from an adjacent nontreated area for comparison.

If herbicide residues in the soil are suspected, certain plant species are better indicators of that herbicide than others. The plants in Table 2 are suggested bioassay species for the corresponding herbicides.

About 10 seeds should be planted per container. Do not plant excess seeds. If too many plants are used, the amount of herbicide in the soil may be diluted.

Injury symptoms on seedlings should become apparent anytime between emergence to three weeks, depending on the herbicide being tested. Water plants sparingly, but do not allow the soil to dry out.

Another way to prevent herbicide resistance is to scout fields regularly to identify resistant weeds. Irregular patches of a single weed species in a field may be an indicator of herbicide resistance, especially when:

- There are no other apparent application problems.
- Other weed species are controlled adequately.
- The weed species that is not controlled shows minimal or no herbicide symptoms.

Table 2. Bioassay Species Used for 10 Selected Herbicides (Washington State University, Cooperative Extension, 1987).

Herbicide	Trade Name	Bioassay Species
Atrazine	Atrazine	Cucumber, oats, wheat, Japanese millet, tomato, pumpkin, pea
Dichlobenil	Casoron 4G	Carrot
Diuron	Diuron 4L, 80DF, 80 WDG	Cucumber, barley, oat, pumpkin, ryegrass
Metolachlor	Pennant Liquid	Japanese millet
Napropamide	Devrinol	Wheat
Oryzalin	Surflan	Oat, barley, wheat
Pronamide	Kerb	Wheat
Simazine	Princep Liquid	Oat, ryegrass, wheat, mustard, sugarbeet, tomato
Trifluralin	Treflan	Oat, barley, annual ryegrass, cucumber
2,4-D	Various	Cucumber, mustard, tomato

- There has been a previous failure with the same herbicide or with a herbicide with the same site of action.
- Records show repeated use of one herbicide or of herbicides with the same site of action (Mallory-Smith *et al.,* 1999).

Another important way to prevent resistance is to rotate crops and change tillage practices. This is the biggest dilemma for weed resistance in nursery production. Often the same crop may be on a field for several years.

Different crops usually require different herbicides; rotating crops can increase herbicide rotation. Although the same crop is on the field in nursery culture for many years, herbicides with different modes of action still need to be rotated. This poses more of a challenge in the nursery; however, it is no less important.

In annual agronomic crops, spring and winter crops may be alternated, meaning the field will be tilled at different times of the year. This is not the case in nursery/perennial crop production; therefore, more reliance should be placed on other non-chemical control methods such as hand weeding, mulching, or solarization of the soil.

Summary

Directed sprays of Glyphosate allow selective removal of weed species in established nursery plantings (Ahrens, 1980, and Haramaki *et al.*, 1980) without crop injury if proper application techniques are used. Preemergent herbicides, however, must be applied following Glyphosate application for residual weed control (Akers *et al.*, 1984).

Achieving satisfactory weed control in field-grown nursery crops generally requires more than one application of herbicides (Gilliam *et al.,* 1989). Spring and fall preemergent sprays are usually recommended.

Four preemergent herbicides that have been commonly used in such programs are Surflan, Princep, Barricade, and Goal. Goal and Princep, however, may cause injury to certain field-grown nursery crops, and so, in recent years, Gallery, a soil-active herbicide, has become more widely used.

Surflan is a nonvolatile herbicide that is primarily active against small-seeded annual broadleaf and grass species. Valor also now has a 24C designation in Ohio for field nursery stock. Valor is recommended for spring or fall application.

Tank mixing two herbicides such as Surflan/Gallery or Gallery/Barricade (no more than 2 to 3 lbs/season) for a fall- or late-summer application is a common nursery-field practice; however, there are some important principles to remember regarding tank mixes and herbicide resistance prevention.

First, the mixture must contain herbicides with different modes of action, which the herbicides listed here do have. Second, combinations that control a broad spectrum of weeds, *i.e.,* a mixture of grass and broadleaf herbicides, may make sense in terms of overall weed control, but these combinations are not a weed-resistance management strategy (Mallory-Smith *et al.,* 1999, and Martin *et al.,* 2001).

The mixture must have herbicides that act on the same weed species to effectively provide multiple modes of action (Martin *et al.,* 2001). This is especially true if a potentially resistant weed is present.

Some tank-mixes should always be avoided whether for overall or resistance management strategies. An example of a combination to avoid is making combinations of contact and systemic activity postemergents. Systemic herbicides are the herbicides of choice for perennial weed control. Although injury symptoms do occur sooner with systemic and contact combinations, perennial regrowth will be quicker (Derr 2001).

Weed escapes and resistance outbreaks are difficult to tell apart. A greenhouse test is required to confirm that a resistant biotype has developed. Weed escapes can be the result of poor herbicide performance.

Poor herbicide performance can be caused by many factors, including improper timing, lack of rain (if needed for activation), poor application coverage, plant stress, failure to use a needed additive, below- or above-usage rates, improper water volume, poor water quality, improper nozzles, and soil type to name a few.

Much time is spent arguing about terminology. Is Johnsongrass resistant to atrazine? Is pigweed resistant to Fusilade? Actually, atrazine has never worked on Johnsongrass, and Fusilade has never worked on pigweed. *Tolerant* would be a better word to describe the response of Johnsongrass and pigweed to atrazine and Fusilade, respectively (Kendig and Fishel, 1996).

Regardless of terminology, the issue today is that weeds that once were controlled are now tolerant to particular herbicides. By adopting proactive management strategies designed to prevent herbicide resistance, we can conserve important weed-control tools (Prather *et al.,* 2000).

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