



Herbicide Resistance on Pennsylvania Roads

The control of weeds along Pennsylvania's roads is an integral part of ongoing management to preserve the integrity and function of the corridor. Several programs use herbicides to control weeds. Nature is resilient though and the chemistry that works today may not tomorrow. This technical bulletin defines herbicide resistance, what it means for the roadside manager, and how to overcome the obstacles faced by this threat.

What is Herbicide Resistance?

When herbicides are applied to a target plant and control is achieved the plant is 'susceptible'. Herbicide susceptibility is the ultimate goal when choosing the herbicide, rate, timing, and application method to target unwanted plants. Sometimes targeted plants do not respond to the chemistry. This can be a result of either herbicide *tolerance* or *resistance*. The distinction between these two must be made before continuing the discussion on herbicide resistance.

Herbicide Tolerance

Tolerance is when a species is not controlled by an herbicide, and never has been. Grasses are not controlled by growth regulator herbicides such as 2,4-D or triclopyr – grasses are tolerant to these herbicides.

Selecting Tolerant Populations

Repeated use of a particular herbicide or combination of herbicides can cause a shift in the weed species present on a site. First use of the selected herbicides most often result in effective control of the target weeds. This visible positive result is largely due to the fact that a vast majority of the species present is susceptible to the herbicide activity. If the same herbicides are used repeatedly over several growing seasons the ratio of susceptible to tolerant species will shift with more tolerant species present in future years. For example, 2,4-D used to control herbaceous species such as dandelion in a bluegrass lawn also containing tall fescue. Dandelion populations are reduced; however, tall fescue thrives due to its tolerance to 2,4-D. In the bluegrass lawn situation, tall fescue becomes the predominant weed.

Consider this scenario in a highway guiderail situation that is treated every year to keep the site free of vegetation. If only a few plants of a tolerant species establish towards the end of the growing season, they often go unnoticed because the overall visible survey is positive. If these

plants complete their life cycle and drop seed; next year there will be more plants. Each year the number of tolerant plants increase until finally the site is heavily populated with tolerant vegetation.

The herbicide combination has not become ineffective because of a loss of activity – instead we have selected or “released” a population of weeds tolerant to the treatment. In southeastern PA, this situation has been observed with the species sprangletop (*Leptochloa fascicularis*) in both industrial and guiderail sites.

Herbicide Resistance

Herbicide resistance can result from repeated use of the same herbicide or combination of herbicides year after year on a target species. The term resistance is used when a species that was originally controlled by a product is no longer controlled at rates that were previously effective. Resistance occurs when there are sub-populations or biotypes within a species that are affected much less by an herbicide than the rest of the population. In time, the repeated use of the herbicide allows the resistant biotype to flourish.

Key Terms to Understand

Table 1. Key terminology for understanding herbicide resistance.

Herbicide families	A method of categorizing herbicides. Herbicides within a family share a similar chemical structure, act on the same sites within the plant, and result in similar injury.
Mode of action	This refers to the metabolic activity within the plant that is impacted by the herbicide.
Site of action	The specific area within the plant that is affected by the herbicide. Some herbicides act on multiple locations within the plant.
Metabolism	A process within the plant that alters the herbicide and reduces the toxic affect.
Biotype	A group of plants within the same species that share a common trait or characteristic that is not found within the rest of the population.
Selection intensity	The extent to which a particular weed control practice or herbicide program allows for the survival of resistant biotypes.

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The Selection or 'Release' of a Resistant Form

Within a given species, there are slightly different genetic populations or biotypes that occur. Each individual in a population is not genetically identical. Sometimes the differences take the form of slightly different structures of the same enzyme. This was the case with biotypes resistant to the triazine herbicides atrazine and simazine. The difference between a susceptible biotype and resistant biotype was a single amino acid substitution in the protein where the triazine herbicide was bound. The resistant biotypes survived and reproduced. In time, the susceptible plants were eliminated and what remained was a growing population of the resistant plants.

Herbicide Behavior in Plants

Herbicides have a difficult route to travel from outside the plant, through its plumbing, finally reaching areas where it acts upon the plant. The herbicides are impacted first by deposition. Deposition is the landing of the herbicide on the intended target. In the case of foliar treatments, the herbicide needs to find itself on the living leaves or stems of the plant. Once on these surfaces it must be drawn through the tissue to the internal parts of the plant. This is referred to as "absorption". Next, the herbicide is translocated or moved through the vascular system of the plant. Ultimately, the herbicide finds its way to the site within the plant where it is designed to disrupt the plant's chemical functions. Slight genetic differences anywhere along the pathway that the herbicide travels may render it ineffective.

Genetic Differences

Within the population of a weed, there is genetic variability. Some of this variability is in the enzymes that occur in one plant compared to another. Enzymes are large, complex molecules which assist in splitting or uniting other building block molecules which form cell walls, important proteins for food production, reproduction, chemical pathways, energy creation, etc. required for maintaining the life of the cells and the whole plant. This variability between forms of the enzyme may not affect how the enzyme works in the cell, but it can affect how an herbicide works.

Herbicides like sulfometuron work by binding to a specific enzyme within the cell and preventing specific amino acid synthesis. This action inhibits the formation of branched chain amino acids necessary in replication of DNA within cells. In other words, it ultimately prevents cell growth in the plant and the plant dies. This enzyme occurs in slightly different forms, and it turns out that sulfometuron and related herbicides can only bind to one of the forms of this enzyme. Repeated use of sulfometuron against kochia resulted in eliminating the plants that had the susceptible form of the enzyme, and increasing the kochia plants that had the resistant form of the enzyme.

Resistance Mechanisms

Plants can make an herbicide ineffective in several ways. Two methods of developing resistant populations include altering the site within the plant where the herbicide is designed to work referred to as 'site of action' or affecting the absorption or translocation of the herbicide in the plant referred to as 'altered metabolism'.

Site of Action

One common mode of action among herbicides is known as amino acid synthesis inhibitors. There are 20 amino acids necessary for creating proteins within plants. Proteins are comprised of 500 to many thousand amino acids that fit together like a perfect puzzle. Each protein is vital to the life and function of the plant. Stopping the production of amino acids, in turn stops protein production and ultimately growth.

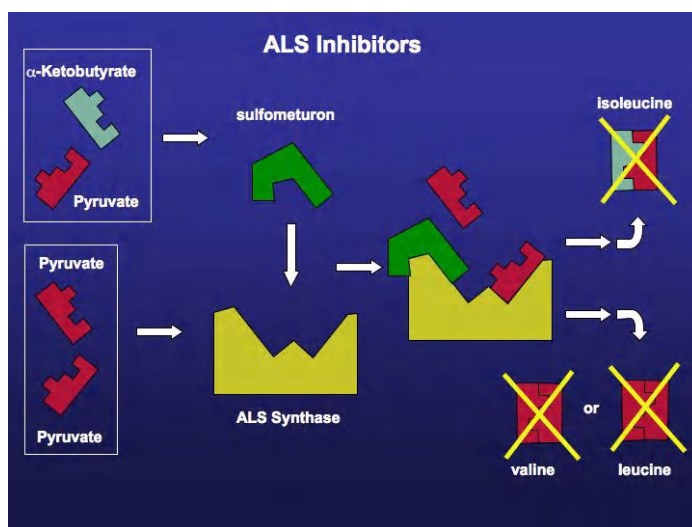


Figure 1. Illustration of the sulfometuron molecule binding with ALS Synthase and preventing the production of three amino acids – isoleucine, leucine, and valine.

Sulfometuron is an amino acid inhibitor. It blocks the pathway for the production of three amino acids known as "isoleucine", "leucine", and "valine". Figure 1 shows the sulfometuron molecule adhering to the enzyme ALS synthase and preventing the amino acids from being constructed.

ALS resistant biotypes or species have ALS synthase enzymes that do not allow the sulfometuron molecule to bind to it. The construction of needed amino acids continues uninterrupted. Sulfometuron and other related herbicides used on these plants do not impact the production of the amino acids. The herbicide is ineffective. This concept is demonstrated in Figure 2.

Altered Metabolism

Marestail is a weed that has demonstrated resistance to glyphosate. The mechanism for resistance of this plant to glyphosate is not in the prevention of amino acids. Translocation or movement of the herbicide through the plant is inhibited by some biotypes of

marestail. This prevents the herbicide from gaining access to the target areas in which it works to control the plant.

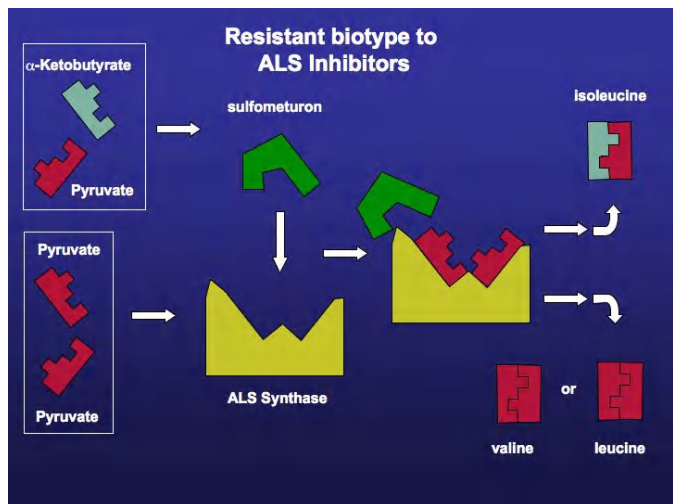


Figure 2. Illustration of a resistant biotype with an ALS synthase enzyme that prevents sulfometuron from binding. The production of the amino acids “isoleucine”, “leucine”, and “valine” is unaffected.

Table 2: Hypothetical growth of a population of herbicide resistant plants. The resistant plants are less vigorous than the susceptible plants, and make up only 0.1 percent of the population initially. For this example, use of the herbicide that the small population is resistant to allows it to double in size each year. After 10 years, the product is ineffective.

Years of Herbicide Use	Resistant Population (%)	Susceptible Population (%)
1	0.1	99.9
2	0.2	99.8
3	0.4	99.6
4	0.8	99.2
5	1.6	98.4
6	3.2	96.8
7	6.4	93.6
8	12.8	87.2
9	25.6	74.4
10	51.2	48.8

This is just another means of herbicide resistance within the plant community.

Vigor of Resistant Biotypes

Some resistant biotypes take time to develop sizeable stands. Such is the case of triazine resistant biotypes. The resistant biotypes were less vigorous, and originally made up a very small portion of the population. It took many years of repeated use of triazine herbicides before the resistant biotypes became apparent. Table 2 illustrates how a resistant biotype making up just 0.1 percent of a species can increase because of repeated use of an herbicide. In this case, it takes eight to nine years before the population shift is noticeable in terms of weed control. Also notice that the visible shift occurs over just a two or three year period, after several years of buildup. This is why many instances of triazine herbicide resistance seemed sudden.

Recently, species in the western part of the US, such as kochia and prickly lettuce have shown resistance to the ‘ALS-inhibitor’ herbicides chlorsulfuron, metsulfuron methyl, sulfometuron methyl, and imazapyr. The resistant biotypes were not less vigorous, and made up a larger portion of the existing population. They were able to increase their population to levels rendering herbicides ineffective much more quickly than the less vigorous biotypes of triazine-resistant weeds. In the case of the ALS-inhibitor herbicides, the weed control failures occurred after a few seasons.

Resistant Species on the Roadside

There are several known resistant biotypes already present in Pennsylvania. Some were discovered as early as 1980. Others have been detected more recently.

Table 3. Known resistant plant ‘biotypes’ found in Pennsylvania and the corresponding failing mode of action.

Weed	Mode of Action
Smooth pigweed	Photosystem II inhibitors
Lambsquarters	Photosystem II inhibitors
Redroot pigweed	ALS & photosystem II inhibitors
Shattercane	ALS inhibitors
Marestail	Glycines
Giant foxtail	ALS inhibitors
kochia	ALS inhibitors

Kochia

Kochia (*Kochia scoparia*) is an annual weed that is becoming more common in guiderail and shoulder areas. This plant is spreading because it is well-adapted to roadside bareground areas, and because the most common ingredient in PennDOT’s herbicide mixture to treat guiderail areas is no longer effective against kochia. The Department relies on the herbicide sulfometuron (an active ingredient in the product Oust Extra) in its bareground herbicide program to control emerged weeds and provide soil residual activity to prevent weed



Figure 3. A mature stand of unwanted kochia growing in guiderail areas within the median.

germination through the season. Within a few years of being introduced in the 1980's, sulfometuron applications stopped being effective against kochia in the Midwest and Plains states. Widespread, repeated use of sulfometuron and closely related chemicals selected for a strain, or biotype, of kochia that was resistant to these herbicides.

Suspected and Potential Resistant Species

The bareground, or 7712 program offers the greatest concern for herbicide resistance. The guiderail and shoulder areas are treated annually in most cases to prevent vegetation from developing. These locations foster annual weed populations that rely on rapid growth and high reproduction rates to sustain themselves. The frequency of treatments, assortment of species, and number of plants creates a tremendous opportunity for the development of resistant biotypes.

Several annual species that are of notable concern in these areas include: common ragweed which has demonstrated resistance to glyphosate; marestalk which has shown resistance to glyphosate, diuron, and herbicides closely related to sulfometuron; and ALS-resistant giant foxtail and kochia.

Reducing Resistance

Herbicide resistance is prevented by rotating herbicide mixtures to assure use of herbicides with different modes of action. In planning your herbicide rotation keep in mind the mode of action of the alternative herbicides to make sure you are changing more than just name brands or formulations. Different

herbicide families can have identical modes of action. For example, replacing sulfometuron (Oust) with imazapyr (Arsenal) would not be effective resistance management. Both herbicides have the exact same mode of action. A biotype that is resistant to Oust may also be resistant to Arsenal.

Most of these mixtures are accomplished by tank mixing several components that have differing modes of action. Some prepackaged herbicides offer a

Table 4. One possible scenario of a three-year rotation for the bareground, 7712 program.

Mix no	products	Product/ac
1	Oust Extra + Karmex XP + Glyphosate	4 oz 8 lb 64 oz
2	Oust Extra + Krovar I + Glyphosate	3 oz 6 lb 64 oz
3	Throttle XP + Glyphosate	12.5 oz 64 oz

combination of active ingredients, such as Sahara DG which contains imazapyr, an amino acid inhibitor, and diuron, a photosystem II inhibitor.

It is not enough to combine various modes of action for a single application. Several effective tank mixes should be developed and rotated to ensure that resistance to a single tank mix does not occur over time. Table 4 outlines an example rotation for use in the bareground, 7712 program. This rotation will help guard against the threat of resistant kochia. Treated areas should also be monitored on a regular basis to watch for the development of weed species that should otherwise be controlled.

Summary

Herbicides are an effective tool for managing roadside vegetation. The concern of developing resistant plant biotypes, particularly in the bareground program, always needs to be a consideration when putting together an effective program. Resistant plants are already here and more will develop in the years ahead. To help minimize the impact and severity of the problem take a proactive approach. Develop several suitable herbicide mixtures, use them in rotation, and monitor for potential misses.

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