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Weed Resistance to Herbicides

Herbicide-resistant weed biotypes continue to plague farmers across much of Illinois. Biotypes are populations within a species that possess characteristics not common to the species as a whole. In this case, the "uncommon characteristic" is resistance to a particular herbicide. Understanding how herbicide resistance develops is an important initial step in designing effective weed-management strategies that deter the selection for resistant biotypes. Table 1 provides a listing of weed species in Illinois that have biotypes resistant to particular herbicide families.

The occurrence of herbicide-resistant weeds has increased during the past decade, but the first reports of herbicide-resistant weeds were documented as early as the 1950s, when dandelion and wild carrot biotypes were reported to be resistant to 2,4-D. Triazine-resistant common groundsel was first reported in 1968 in Washington. Worldwide, more than 183 weed species have been reported to possess resistance to one family of herbicides or another.

The terminology used when discussing herbicide resistance can be confusing. The most common terms are defined as follows:

Herbicide resistance: Resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type.

Herbicide tolerance: Tolerance is the inherent ability of a species to survive and reproduce after herbicide treatment.

Let's examine these definitions more closely. Notice in the definition of resistance, the word "plant" is used, whereas "species" is used in the definition of tolerance. Stated another way, a resistant plant is a member of a species that, as a whole, is susceptible to the herbicide. The resistant plant is a **biotype** of that species that is no longer susceptible to the herbicide. Tolerance implies the species has never been susceptible to the herbicide.

Other terms related to herbicide resistance include the following:

Cross-resistance: Resistance to a herbicide the plant may not have been previously exposed to but that has a mode or site of action similar to the original herbicide.

Multiple-resistance: Resistance to more than one class of herbicides with very different modes or sites of action in which more than one basis for resistance may be involved.

The following examples may help to eliminate confusion about these terms. A producer who has grown continuous corn on the same field for many years has used atrazine (a photosynthesis-inhibiting herbicide) each year for weed control. He or she notices that in recent years the control of common lambsquarters has been poor. The local Extension educator collects seed from the common lambsquarters and, during the winter, confirms that the weed is **resistant** to atrazine. The producer then decides to switch to simazine (another photosynthesis inhibitor) the following year and again finds the control of common lambsquarters to be poor. Further investigation reveals that the common lambsquarters is also resistant to simazine. Because the plants are resistant to both atrazine and simazine, they are said to exhibit **cross-resistance**. The next year, the producer decides to use a post-emergence application of Clarity (a growth-regulating herbicide) to control the common lambsquarters, and

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Label registrations can change at any time. Thus the recommendations in this chapter may become invalid. The user must read carefully the entire, most recent label and follow all directions and restrictions. Purchase only enough pesticide for the current growing season.

once again poor control results. Investigations reveal that the common lambsquarters is also resistant to Clarity, a situation that is defined as **multiple-resistance**. A documented example of multiple-resistance is a biotype of waterhemp from western Illinois. This biotype has demonstrated resistance to such herbicide families as the acetolactate synthase (ALS) inhibitors, triazines (atrazine, simazine), and protoporphyrinogen oxidase (PPO) inhibitors. If these forms of resistance were ranked from least difficult to most difficult to control with herbicides, the order would be resistant < cross-resistant < multiple-resistant.

Origin of Resistance

To slow the selection of herbicide-resistant weeds, one should have a basic understanding of how a resistant weed population develops. Two mechanisms have been proposed: the mutation theory and the natural-selection theory.

The mutation theory postulates that a genetic mutation occurs within a plant following the application of a herbicide and that this mutation confers resistance to the plant. There is little evidence to support this theory, and it is disregarded by most scientists as a valid explanation for the development of resistance to herbicides.

The natural-selection theory is widely regarded as the most plausible explanation for the development of resistance. The theory states that herbicide-resistant biotypes have always existed at extremely low numbers within particular weed species. When a herbicide effectively controls the majority of susceptible members of a species, only those plants that possess a resistance trait can survive and produce seed for future generations.

This theory of resistance development has several parallels to Darwin's theory of survival of the fittest. Biological organisms (humans, plants, animals, etc.) exhibit a wide range of diversity. No two people are exactly the same, and plants likewise show extreme diversity. The plants that are in a population with characteristics enabling them to survive under a wide range of environmental and other adverse conditions will be the ones to produce seed that maintains these survival characteristics. The plants less adapted do not survive, and hence only the fittest plants produce seed. Plants that possess characteristics (such as resistance to herbicides) that are not common to the entire species are referred to as "biotypes." The characteristics possessed by resistant biotypes that confirm herbicide resistance will be presented later in the chapter.

What then is meant by "selection pressure" in regard to herbicide-resistant weeds? Herbicides are used to control a wide spectrum of weeds. By controlling susceptible members of a weed population, we are essentially using herbicides as agents to select for biotypes that are naturally resistant to the herbicide. When most of the susceptible members of a weed population are controlled, the resistant biotypes are able to continue growing and eventually produce seed. The seed from the resistant biotypes ensures that the resistance trait carries into future seasons. If the same herbicide is used year after year, or several times during a single season, the resistant biotypes continue to thrive, eventually outnumbering the normal (susceptible) population. In other words, relying on the same herbicide (or herbicides with the same site of action) for weed control creates selection pressure that favors the development of herbicide-resistant weeds.

The development of a herbicide-resistant weed population can be summarized by the following principle: *The appearance of herbicide-resistant weeds is the consequence of using a herbicide with a single site of action year after year or of repeating applications of a herbicide during the growing season to kill a specific weed species not controlled by any other herbicide or in any other manner.* This principle has three key components:

1. A herbicide with a single site of action.
2. Repeated use of the same herbicide.
3. The absence of other control measures.

By understanding these components and developing weed-control systems with them in mind, producers can greatly reduce the probability that herbicide-resistant weeds will develop in their fields.

Basis for Weed Resistance

What occurs within a resistant plant that allows it to survive after a herbicide application? What characteristics do the resistant plants possess that the susceptible plants lack? Two mechanisms have been identified that account for the majority of observed cases of herbicide resistance:

1. **Alterations in the target site of the herbicide**. A herbicide has a specific site within the plant where it acts to disrupt a particular plant process or function. If this target site is somewhat altered, the herbicide molecule may be unable to exert its phytotoxic action effectively. Thus far, most cases of herbicide resistance have involved alterations in the herbicide target site. Examples include resistance to triazine (atrazine, simazine, and others), ALS-inhibiting herbicides (imazaquin, chlorsulfuron, and others), and ACCase-inhibiting herbicides (sethoxydim, fenoxaprop, and others).

2. **Enhanced metabolism of the herbicide**. Metabolism within the plant is one mechanism a plant uses to detoxify a foreign compound such as a herbicide. A weed with an enhanced ability to metabolize a herbicide can potentially inactivate it before it can reach its site of action within the plant. A triazine-resistant biotype of velvetleaf from Maryland has been identified that possesses an enhanced ability to metabolize the herbicides atrazine and simazine. Generally, as stated earlier, weed resistance to triazine herbicides is attributed to alterations in the target site of the herbicide. This velvetleaf biotype, however, possesses an enhanced enzyme activity that rapidly metabolizes the herbicide to nonphytotoxic forms.

Management Strategies to Minimize Herbicide-Resistant Weeds

The best solution for minimizing herbicide-resistant weeds is to prevent their selection. In the past, as new weed problems were discovered, the usual solution has been to develop new herbicides. Today, the high cost of developing a new herbicide makes good management practices the best method for dealing with herbicide-resistant weeds. The following management strategies may help deter the development of herbicide resistance:

- Scout fields regularly to identify resistant weeds. Respond quickly to changes in weed populations to restrict the spread of plants that may have developed resistance.
- Rotate herbicides with different sites of action. Do not make more than two consecutive applications of herbicides with the same site of action against the same weed unless other effective control practices are included in the management system. Consecutive applications can be single applications in 2 years or two split applications in 1 year.
- Apply herbicides in tank-mixed, prepackaged, or sequential mixtures that include multiple sites of action. Both herbicides in the mixture must have substantial activity against potentially resistant weeds, as well as similar soil persistence.
- As new herbicide-resistant and herbicide-tolerant crops become available, their use should still not result in more than two consecutive applications of herbicides with the same site of action against the same weed unless other effective practices are included in the management system.
- Combine mechanical control practices (such as rotary hoeing, cultivating, and even hand weeding) with herbicide treatments for a near-total weed-control program.
- Clean tillage and harvest equipment before moving from fields infested with resistant weeds to fields that are not infested.
- Railroads, public utilities, highway departments, and similar organizations using total-vegetation-control programs should be encouraged to use practices that do not lead to the development of herbicide-resistant weeds. Resistant weeds resulting from areas of total vegetation control frequently spread to cropland. Chemical companies, state and federal agencies, and farm organizations can help in this effort.

Several criteria may be used to diagnose a herbicide-resistant weed problem correctly:

- All other causes of herbicide failure have been eliminated.
- Other weeds on the herbicide label (besides the one in question) were controlled effectively.
- The field has a history of continuous or repeated use of the same herbicide or herbicides with the same site of action.
- The weed species was controlled effectively in the past. Weed control in the field has been based entirely on herbicides without mechanical control.

With these management strategies and diagnosis criteria in mind, how does one go about correctly identifying a resistant weed population? We know that initially resistant weed biotypes are present at extremely low frequencies within a particular population. It stands to reason, then, that because of such a low initial frequency, resistance will most likely be first noticed within a particular field as a few individual weeds that were not controlled. In other words, resistant weeds do not usually infest an entire field within 1 year. Typically, the resistant weed population is initially confined to small, isolated patches. If the same herbicide-control program is followed repeatedly, these patches begin to encompass a larger proportion of the field until finally the resistant weeds appear as the dominant species. So a producer who encounters an entire field of resistant weeds has most likely had a resistant population in the field for more than 1 year.

How can the spread of resistant weeds be confined? Early identification of the problem, using the information provided in this chapter, ultimately proves beneficial. A hypothetical scenario may help put all these pieces of the resistance puzzle together.

A producer has grown continuous corn for the last 10 years on a particular 40-acre farm, using atrazine at the highest allowable rate each year to control broadleaf weeds. While scouting this field during the growing season, the producer notices several lambsquarters in a small patch (say 30 feet in diameter) but observes that all other weed species commonly encountered in this field were effectively controlled. The producer knows that atrazine has been used continuously on this field for 10 years and realizes that, because all other weeds that are susceptible to atrazine were controlled, this may be the early stages of the development of a triazine-resistant population of lambsquarters. With this in mind, the producer eradicates the small patch of lambsquarters by hand hoeing so that no seed will be produced by those plants. Needless to say, the producer should develop an alternative weed-management program for future years that does not rely exclusively on triazine herbicides.

Tables 2 and 3 list herbicides and herbicide premixes according to their respective sites of action. Table 2 further divides the herbicides into those that possess higher or lower potential to select for resistant weeds. The classifications are based primarily on two criteria: how extensively a particular herbicide active ingredient is (or has been) used in Illinois and scientific documentation of resistance to a particular herbicide or herbicide site of action.

Table 1. Weed species in Illinois that include herbicide-resistant biotypes and the herbicide families to which these biotypes are resistant

Weed species		
Common name	Scientific name	Resistant to herbicide family(ies)
common lambsquarters	*Chenopodium album*	triazine
smooth pigweed	*Amaranthus hybridus*	triazine, ALS inhibitors
kochia	*Kochia scoparia*	triazine, ALS inhibitors
common waterhemp	*Amaranthus rudis*	triazine, ALS inhibitors, PPO inhibitors, glyphosate
eastern black nightshade	*Solanum ptycanthum*	ALS inhibitors
giant ragweed	*Ambrosia trifida*	ALS inhibitors
common ragweed	*Ambrosia artemisiifolia*	ALS inhibitors
common cocklebur	*Xanthum strumarium*	ALS inhibitors
shattercane	*Sorghum bicolor*	ALS inhibitors
giant foxtail	*Setaria faberi*	ACCase inhibitors, ALS inhibitors
horseweed	*Conyza canadensis*	glyphosate

Table 2. Resistance potential of herbicides according to site of action

Higher potential	Lower potential
Inhibitors of acetyl-CoA carboxylase (ACCase)	**Inhibitors of microtubule assembly**
Aryloxyphenoxy propionates	*Dinitroanilines*
fenoxaprop (Puma)	benefin (Balan)
fluazifop (Fusilade DX)	pendimethalin (Prowl, Pendimax)
quizalofop (Assure II)	trifluralin (Treflan, others)
Cyclohexanediones	**Synthetic auxins—specific site unknown**
clethodim (Select, Select Max)	*Phenoxys*
sethoxydim (Poast Plus)	2,4-D (Weedone, others)
Inhibitors of acetolactate synthase (ALS)	MCPA (various)
Sulfonylureas	MCPP (various)
chlorimuron (Classic)	*Benzoic acids*
chlorsulfuron (Telar)	dicamba (Banvel, Clarity, Status)
foramsulfuron (Option)	*Carboxylic acids*
halosulfuron (Permit)	clopyralid (Stinger)
metsulfuron (Cimarron)	fluroxypyr (Starane)
nicosulfuron (Accent)	picloram (Tordon)
primisulfuron (Beacon)	triclopyr (Garlon)
prosulfuron (Peak)	**Inhibitors of Photosystem I**
rimsulfuron (Resolve)	*Bipyridiliums*
sulfometuron (Oust)	diquat (Reward)
thifensulfuron (Harmony GT XP)	paraquat (Gramoxone Inteon)
tribenuron (Express)	**Inhibitors of EPSP synthase**
Imidazolinones	glyphosate (Roundup, Touchdown, others)
imazamox (Raptor)	**Inhibitors of glutamine synthetase**
imazapic (Cadre, Plateau)	glufosinate (Liberty)
imazapyr (Arsenal)	**Inhibitors of lipid biosynthesis, not via ACCase**
imazaquin (Scepter)	*Thiocarbamates*
imazethapyr (Pursuit)	butylate (Sutan+)
Triazolopyrimidines	EPTC (Eradicane)
cloransulam (FirstRate)	**Bleaching: Inhibitors of diterpene synthesis**
flumetsulam (Python)	*Isoxazolidinones*
Inhibitors of photosynthesis at Photosystem II	clomazone (Command)
Triazines	**Bleaching: Inhibitors of 4-HPPD**
ametryn (Evik)	*Isoxazoles*
atrazine (AAtrex, others)	isoxaflutole (Balance Pro)
prometon (Pramitol)	mesotrione (Callisto)
simazine (Princep)	topramezone (Impact)
Triazinones	
hexazinone (Velpar)	
metribuzin (Sencor)	
Uracils	
bromacil (Hyvar)	
terbacil (Sinbar)	

Table 2. Resistance potential of herbicides according to site of action (cont.)

Higher potential	Lower potential
Inhibitors of photosynthesis at Photosystem II —same site, different binding behavior	**Inhibitors of photosynthesis at Photosystem II —same site, different binding behavior**
Ureas	*Nitriles*
diuron (Karmex, Direx)	bromoxynil (many)
linuron (Lorox)	
tebuthiuron (Spike)	*Benzothiadiazoles*
	bentazon (Basagran)
Inhibitors of protoporphyrinogen oxidase	
Diphenylethers	**Unknown**
acifluorfen (Ultra Blazer)	*Chloroacetamides*
fomesafen (Flexstar, Reflex)	acetochlor (Degree, Harness, TopNotch)
lactofen (Cobra, Phoenix)	alachlor (IntRRo, Micro-Tech, Partner)
	dimethenamid (Outlook)
N-phenylphthalimides	*S*-metolachlor (Dual Magnum, Dual II Magnum)
flumiclorac (Resource)	
flumioxazin (Valor)	*Oxyacetamides*
	flufenacet (Define)
Aryl triazinones	
carfentrazone (Aim)	
sulfentrazone (Spartan)	

Table 3. Premix herbicides with at least one herbicide component with a high potential for contributing to weed resistance

Photosynthetic inhibitors	ALS inhibitors
atrazine Bicep Lite II Magnum (atrazine + *S-metolachlor*)* Bicep II Magnum (atrazine + *S-metolachlor)* Buctril + atrazine (atrazine + bromoxynil) Bullet (atrazine + *alachlor)* Degree Xtra (atrazine + *acetochlor)* Expert (atrazine + *S-metolachlor* + *glyphosate)* FieldMaster (atrazine + *acetochlor* + *glyphosate)* FulTime (atrazine + *acetochlor)* Guardsman Max (atrazine + *dimethenamid-P)* Harness Xtra (atrazine + *acetochlor)* Keystone (atrazine + *acetochlor)* Keystone LA (atrazine + *acetochlor)* Laddok S-12 (atrazine + *bentazon)* Lexar (atrazine + *S-metolachlor* + *mesotrione)* Lumax (atrazine + *S-metolachlor* + *mesotrione)* Marksman (atrazine + *dicamba)* Shotgun (atrazine + *2,4-D)* Steadfast ATZ (atrazine + *nicosulfuron* + *rimsulfuron)* **metribuzin** Boundary (metribuzin + *S-metolachlor)* Canopy (metribuzin + *chlorimuron)*	**imazethapyr** Extreme (imazethapyr + *glyphosate)* Lightning (imazethapyr + imazapyr*)* Pursuit Plus (imazethapyr + *pendimethalin)* **chlorimuron** Canopy EX (chlorimuron + tribenuron) Canopy (chlorimuron + *metribuzin)* Synchrony XP (chlorimuron + thifensulfuron) **cloransulam** Sonic, Authority First (cloransulam + *sulfentrazone)* **flumetsulam** Hornet (flumetsulam + *clopyralid)* SureStart (flumetsulam + *acetochlor* + *clopyralid)* **foramsulfuron** Equip (foramsulfuron + iodosulfuron) **thifensulfuron** Basis (thifensulfuron + rimsulfuron) Harmony Extra (thifensulfuron + tribenuron) Synchrony XP (thifensulfuron + chlorimuron) **primisulfuron** Northstar (primisulfuron + *dicamba)* Spirit (primisulfuron + prosulfuron) **rimsulfuron** Basis (rimsulfuron + thifensulfuron) Steadfast (rimsulfuron + nicosulfuron) Steadfast ATZ (rimsulfuron + nicosulfuron + *atrazine)* **nicosulfuron** Celebrity Plus (nicosulfuron + *dicamba* + *diflufenzopyr)* Steadfast (nicosulfuron + rimsulfuron) Steadfast ATZ (nicosulfuron + rimsulfuron + *atrazine)*

*Herbicides in italics have a different site of action. For example, Bicep Lite II Magnum contains a triazine component (atrazine) and a nontriazine component *(S-metolachlor)*.

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