

WEED RESISTANCE TO HERBICIDES

A dilemma facing producers in many states is the development of biotypes of weed species that are resistant to herbicides. Although the problem of herbicide-resistant weeds is not as widespread in Illinois as it is in some other states, resistance is an issue that should be addressed in this state so producers will know how resistance develops and how to minimize the occurrence of herbicide-resistant weeds.

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; and, to date, resistance to triazine herbicides has been documented most frequently. About 100 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. Several of the most common terms are defined as follows:

Tolerance: Survival of the normal population of a species following treatment with a herbicide dosage lethal to other species.

Resistance: Survival of a segment of the population following treatment with a herbicide dosage lethal to the normal population.

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

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

The following examples may further help to eliminate confusion about these terms. A producer who has grown continuous corn on the same field for many years has used AAtrex (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 adviser collects seed from the common lambsquarters and, during the winter, confirms that the weed is **resistant** to AAtrex. The producer then decides to switch to Bladex (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 Bladex. Because they are resistant to both AAtrex and Bladex, the plants are said to exhibit **cross-resistance**. The next year, the producer decides to use a postemergence application of Banvel (a growth-regulating herbicide) to control the common lambsquarters, and once again poor control results. Investigations reveal that the common lambsquarters is also resistant to Banvel, a situation that is defined as **multiple-resistance**. A documented example of multiple-resistance is rigid ryegrass in Australia. This species has shown resistance to such herbicide families as the aryloxyphenoxypropionates (Fusilade DX, Option II, Assure II) and the sulfonyleureas (Classic, Pinnacle, and others). 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.

The information in this chapter is provided for educational purposes only. Product trade names have been used for clarity, but reference to trade names does not imply endorsement by the University of Illinois; discrimination is not intended against any product. The reader is urged to exercise caution in making purchases or evaluating product information.

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.

ORIGIN OF RESISTANCE

To avoid the development 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 weed resistance.

The natural-selection theory is widely regarded as the most plausible explanation for the development of weed resistance. The theory states that herbicide-resistant weeds have always occurred 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 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 effectively 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, those resistant biotypes are able to continue growing and eventually to 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 mode 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.

So why haven't herbicide-resistant weeds become as prevalent in Illinois as in some other states? Most of the acreage in Illinois is in some type of crop rotation from year to year. Crop rotation often allows for the use of herbicides that have very different modes of action instead of relying on one particular herbicide in a monoculture. Many Illinois producers also incorporate some type of nonchemical control practice (such as cultivation) into their weed control program. However, the development of herbicide-resistant or herbicide-tolerant crops that allow the same herbicide to be used on both corn and soybeans is a cause for concern because it has the potential to expedite the development of herbicide-resistant weeds. In the past, as new weed problems were discovered, the usual solution had been to develop new herbicides. Today, the high cost of developing a new herbicide makes good management practices the best method of dealing with herbicide-resistant weeds.

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? Three mechanisms have been identified that account for the 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 phyto-

toxic action effectively. Thus far, most cases of herbicide resistance have involved alterations in the herbicide target site. Examples include resistance to triazine (atrazine, cyanazine, and others), ALS (imazaquin, chlorsulfuron, and others), and ACCase (sethoxydim, fenoxaprop, and others) herbicides.

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.
3. **Compartmentation of the herbicide.** Plants are capable of sequestering foreign compounds within their cells or tissues to prevent the compounds from causing harmful effects. When a herbicide is placed within a restricted compartment, it cannot reach its site of action and thus is unable to kill the plant. About 18 weed biotypes show resistance to the bipyrilidilium herbicides paraquat and diquat. One of the proposed mechanisms for this resistance is that the resistant biotypes restrict the movement of the herbicides within themselves and do not allow the herbicides to reach their site of action within the chloroplast.

MANAGEMENT STRATEGIES TO MINIMIZE HERBICIDE-RESISTANT WEEDS

The best solution for minimizing herbicide-resistant weeds is to prevent their development. The following management strategies may 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 modes of action. Do not make more than two consecutive applications of herbicides with the same mode of action against the same weed unless other effective con-

trol 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 modes of action. Both herbicides in the mixture must have substantial activity against potentially resistant weeds, as well as similar 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 mode 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 total-vegetation-control areas 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 use of the same herbicide or herbicides with the same mode of action.
- The weed species was controlled effectively in the past. The control in the field has been based 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 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 effectively controlled. In other words, resistant weeds do not 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 an increasingly large 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 broad-leaf 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 continu-

ously 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 1 and 2 list herbicides and herbicide premixes according to their respective modes of action. Table 1 further divides the herbicides into those that possess high and low potential to contribute to the development of resistant weeds. The classifications are based primarily on two criteria: how extensively a particular herbicide active ingredient is used in Illinois, and scientific documentation of resistance to a particular herbicide or herbicide mode of action. These criteria were selected because the only documented cases of resistance in Illinois are triazine- and ALS-resistant weed biotypes, yet the prevalence of weed resistance to other herbicide families has dramatically increased across the country over the past several years.

Table 1. Resistance Potential of Herbicides According to Mode of Action

High potential	Low potential
<p>Photosynthetic inhibitors</p> <p><i>Triazines</i> atrazine (AAtrex, others) cyanazine (Bladex) metribuzin (Sencor/Lexone) prometon (Pramitol) simazine (Princep)</p> <p><i>Phenylureas</i> diuron (Karmex) linuron (Lorox) tebuthiuron (Spike)</p> <p><i>Bipyridiliums</i> diquat (Reward) paraquat (Gramoxone Extra)</p> <p><i>Uracils</i> bromacil (Hyvar) terbacil (Sinbar)</p> <p>Amino acid–synthesis inhibitors</p> <p><i>ALS inhibitors</i></p> <p><i>Imidazolinones</i> imazamox (Raptor) imazapyr (Arsenal, Contain) imazaquin (Scepter) imazethapyr (Pursuit)</p> <p><i>Sulfonylureas</i> chlorimuron (Classic) chlorsulfuron (Telar) halosulfuron (Battalion, Permit) metsulfuron (Ally, Escort) nicosulfuron (Accent) oxasulfuron (Expert) primisulfuron (Beacon) prosulfuron (Peak) sulfometuron (Oust) thifensulfuron (Pinnacle) tribenuron (Express)</p> <p><i>Triazolopyrimidine sulfonanilides</i> cloransulam (FirstRate) flumetsulam (Python)</p>	<p>Photosynthetic inhibitors</p> <p><i>Benzothiadiazoles</i> bentazon (Basagran)</p> <p><i>Phenylpyridazines</i> pyridate (Tough)</p> <p><i>Benzonitriles</i> bromoxynil (Buctril)</p> <p>Amino acid–synthesis inhibitors</p> <p><i>Amino acid derivatives</i></p> <p><i>Phosphonos</i> glyphosate (Roundup, Roundup Ultra) glyphosate trimesium (Touchdown 5)</p>

Table 1. Resistance Potential of Herbicides According to Mode of Action (cont.)

High potential	Low potential
<p>Lipid-synthesis inhibitors</p> <p><i>ACCase inhibitors</i></p> <p><i>Aryloxyphenoxypropionic acids</i> fenoxaprop (Option II) fenoxaprop + fluazifop (Fusion) fluazifop (Fusilade DX) quizalofop (Assure II)</p> <p><i>Cyclohexanediones</i> clethodim (Select) sethoxydim (Poast, Poast Plus, Prestige)</p>	<p>Shoot inhibitors</p> <p><i>Chloroacetamides</i> acetochlor (Harness, Surpass, TopNotch) alachlor (Micro-Tech, Partner) dimethenamid (Frontier) metolachlor (Dual II Magnum) propachlor (Ramrod)</p> <p><i>Thiocarbamates</i> butylate (Sutan +) EPTC (Eradicane)</p> <p>Microtubule disruptors</p> <p><i>Dinitroanilines</i> benefin (Balan) ethalfluralin (Curbit, Sonalan) pendimethalin (Pentagon, Prowl) trifluralin (Treflan, Tri-4)</p> <p>Growth-hormone herbicides</p> <p><i>Phenoxyacetic acids</i> 2,4-D (many) 2,4-DB (Butyrac) MCPA (many)</p> <p><i>Benzoic acids</i> dicamba (Banvel, Clarity, Vanquish)</p> <p><i>Pyridinecarboxylic acids</i> clopypalid (Stinger) picloram (Tordon) triclopyr (Garlon)</p> <p>Carotenoid-synthesis inhibitors</p> <p><i>Isoxazolidinones</i> clomazone (Command)</p> <p><i>Isoxazoles</i> isoxaflutole (Balance)</p> <p>PPO inhibitors</p> <p><i>Diphenyl ethers</i> acifluorfen (Blazer, Status) fomesafen (Flexstar, Reflex) lactofen (Cobra)</p> <p><i>N-phenylthalamides</i> flumiclorac-pentyl (Resource)</p> <p><i>Aryl triazolinones</i> sulfentrazone (Authority)</p> <p><i>Other</i> fluthiacet-methyl (Action)</p>

Table 2. Premix Herbicides with at Least One Herbicide Component with a High Potential for Contributing to Weed Resistance

Photosynthetic inhibitors	ALS inhibitors
<p>atrazine Bicep II Magnum (atrazine + <i>metolachlor</i>*) Bicep Lite II Magnum (atrazine + <i>metolachlor</i>) Bucril + atrazine (atrazine + bromoxynil) Bullet (atrazine + <i>alachlor</i>) Contour (atrazine + <i>imazethapyr</i>) Extrazine II (atrazine + cyanazine) FulTime (atrazine + <i>acetochlor</i>) Guardsman (atrazine + <i>dimethenamid</i>) Harness Xtra (atrazine + <i>acetochlor</i>) Laddok S-12 (atrazine + <i>bentazon</i>) Marksman (atrazine + <i>dicamba</i>) Shotgun (atrazine + 2,4-D) Surpass 100 (atrazine + <i>acetochlor</i>)</p> <p>cyanazine Extrazine II (cyanazine + atrazine)</p> <p>metribuzin Axiom (metribuzin + <i>fluthiamide</i>) Canopy (metribuzin + <i>chlorimuron</i>) Turbo (metribuzin + <i>metolachlor</i>)</p>	<p>imazethapyr Contour (imazethapyr + <i>atrazine</i>) Lightning (imazethapyr + imazapyr) Passport (imazethapyr + <i>trifluralin</i>) Pursuit Plus (imazethapyr + <i>pendimethalin</i>) Resolve (imazethapyr + <i>dicamba</i>) Steel (imazethapyr + imazaquin + <i>pendimethalin</i>)</p> <p>chlorimuron Canopy (chlorimuron + <i>metribuzin</i>) Canopy XL (chlorimuron + <i>sulfentrazone</i>) Concert (chlorimuron + thifensulfuron) Synchrony STS (chlorimuron + thifensulfuron)</p> <p>flumetsulam Accent Gold (flumetsulam + <i>clopyralid</i> + nicosulfuron + rimsulfuron) Broadstrike + Dual (flumetsulam + <i>metolachlor</i>) Broadstrike + Treflan (flumetsulam + <i>trifluralin</i>) Hornet (flumetsulam + <i>clopyralid</i>) Scorpion III (flumetsulam + <i>clopyralid</i> + 2,4-D)</p> <p>imazaquin Detail (imazaquin + <i>dimethenamid</i>) Scepter O.T. (imazaquin + <i>acifluorfen</i>) Squadron (imazaquin + <i>pendimethalin</i>) Steel (imazethapyr + imazaquin + <i>pendimethalin</i>) Tri-Scept (imazaquin + <i>trifluralin</i>)</p> <p>thifensulfuron Basis (thifensulfuron + rimsulfuron) Concert (thifensulfuron + chlorimuron) Harmony Extra (thifensulfuron + tribenuron) Synchrony STS (thifensulfuron + chlorimuron)</p> <p>primisulfuron Exceed, Spirit (primisulfuron + prosulfuron)</p> <p>rimsulfuron Accent Gold (rimsulfuron + flumetsulam + nicosulfuron + <i>clopyralid</i>) Basis (rimsulfuron + thifensulfuron) Basis Gold (rimsulfuron + nicosulfuron + <i>atrazine</i>)</p>

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

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