AGRICULTURAL
MU Guide
PUBLISHED BY UNIVERSITY EXTENSION, UNIVERSITY OF MISSOURI-COLUMBIA

Herbicide Resistance in Weeds

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In 1992 no known herbicide-resistant weeds existed in Missouri. In 1994 there were more than five sites. Herbicide-resistant weeds are populations that were **previously controlled** by a particular herbicide but **are no longer controlled by a normal rate of application for that herbicide**. Much time is spent arguing about terminology: Is johnsongrass resistant to atrazine? Is pigweed resistant to Assure, Bugle, Fusilade, Poast and Select? Johnsongrass always has been resistant to atrazine, and, likewise, pigweed always has been resistant to grass herbicides. Is "tolerant" a better word to describe johnsongrass's response to atrazine? **Regardless of the terminology used, the problem this guide addresses is weeds that used to be controlled but are now tolerant to particular herbicides**.

Herbicide-resistant weeds were first discovered in the United States in the late 1960s in a pine nursery where triazine herbicides had been used repeatedly. There probably have been a few resistant weeds in Missouri over past years, but the first confirmed discovery of a problem did not occur until 1993. By 1994 Missouri had five cases of herbicide-resistant weeds.

Weed resistance is not unique. Insecticide-resistant insects, fungicide-resistant diseases and antibiotic-resistant bacteria were discovered long before herbicide-resistant weeds. These pests have two common traits: They have exceptionally large populations, and they reproduce rapidly. The reason that weeds were the last pest category to show resistance is that they reproduce (set seed) only once a year while insects reproduce several times a year. Some bacteria and fungi reproduce several times in a single hour. Exceptionally large populations also result in a wide variety of types, or genetic diversity, within the population. With most control measures (insecticides, herbicides or antibiotic drugs), small groups in the pest population (one in a million, billion, etc.) are tolerant.

Weeds typically become resistant to herbicides when the same herbicide is used repeatedly for several (4 to 10) years in a row. It often is thought that selection pressure forces the weeds to mutate and become resistant. However, biologists tell us that what most likely happens is (1) There were always a few of the resistant types present. (2) When the herbicide is used, the susceptible types are controlled, and then the smaller, resistant populations increase and reinfest the area.

Resistance makes some weeds less fit than normal weeds. However, other resistant weeds are just as vigorous as normal types, For example, triazine-resistant pigweed has a different photosynthesis metabolism, which is less efficient than that of the normal type. The "mutant" pigweed grows much slower than normal pigweed. But when atrazine is used, the normal pigweed is controlled, and the resistant pigweed thrives because of lessened competition. However, ALS-herbicide-resistant kochia grows just as fast and just as tall as normal kochia.

Look carefully for resistance

How to identify herbicide resistance: As farmers learn about herbicide resistance, an unfortunate side effect is that some herbicide failures from bad weather, weeds that are too large or improper applications are considered herbicide-resistance problems. Do not suspect herbicide resistance unless a herbicide failure fits the following traits:

- The same herbicide was used year after year.
- One weed, which normally should be controlled, is not controlled although other weeds are controlled.
- A patch of an uncontrolled weed is spreading.
- Healthy weeds are mixed with controlled weeds (of the same species).

Even if a control failure exhibits these traits, it is not an absolute diagnosis of herbicide resistance.

Resistance in Missouri and nearby states

ALS-resistant cocklebur. Several newer herbicides target the acetolactate synthase (or ALS) enzyme, which is found only in plants. A Scepter-resistant cocklebur was originally reported in Mississippi in 1993. This cocklebur also showed resis-

tance to herbicides that are chemically related to Scepter (imidazolinone family), such as Arsenal, Cadre and Pursuit. Sulfonylurea (pronounced sulfoneal-urea) herbicides, which also work by inhibiting ALS, were still effective against this cocklebur. At about the same time, a Missouri farmer also was failing to control cocklebur with Scepter. After the grower contacted the University of Missouri, research determined that his cocklebur was **cross resistant** to chemicals from both the imidazolinone and the sulfonylurea families of chemistry. It is noteworthy that this cocklebur was completely resistant to Classic, even though it had never been sprayed in the field, and was completely resistant to Broadstrike (another ALS inhibitor) before it was registered and sold.

ACCase-resistant johnsongrass, crabgrass. The postemergence grass herbicides (Assure, Bugle, Fusilade, Option, Poast and Select) inhibit an enzyme called acetyl coenzyme A carboxylase, or "ACCase." In 1993 johnsongrass resistant to postemergence grass herbicides was reported in Mississippi. In subsequent research, the johnsongrass tolerated normal-use rates of Assure, Fusilade and Poast. Labeled rates of Select were still killing the grass, but detailed studies showed that the grass was more resistant to low Select rates than was normal johnsongrass. Crabgrass with similar resistance has been found in the northern United States. At the time of this publication, resistant johnsongrass also has been found in Arkansas, but not in Missouri.

Glean-resistant prickly lettuce/kochia. The first cases of ALS-resistant weeds involved prickly lettuce, kochia and the wheat herbicide Glean. Glean made headlines as the first of the "ounce-per-acre" herbicides. However, after four to five years of use, growers began to observe prickly lettuce and kochia escaping Glean treatments. After four additional years, Glean-resistant weeds were widespread across the western United States.

Londax-resistant weeds — California rice. The rice herbicide Londax is another sulfonylurea, ALS-inhibiting herbicide. Approximately four years after its labeling, a resistant arrowhead was found. Recent estimates indicate that one in three California rice fields have at least one Londax-resistant weed.

MSMA/DSMA-resistant cocklebur. DSMA and MSMA are closely related, arsenical herbicides that are commonly used in cotton and turfgrass. DSMA and MSMA are used primarily for grass control although they also provide a significant amount of broadleaf suppression. Although they often are considered "grass herbicides," they have good activity on cocklebur and are a particularly economical option for its control. Resistant cocklebur is difficult to identify in a field setting because the resistant types are injured by DSMA and MSMA just as the normal types of cocklebur are injured. The resistant types recover, however, and the susceptible types die. Resistant cocklebur has been discovered in cotton fields in Arkansas, Louisiana, Mississippi and Tennessee. At the time this guide was prepared, a preliminary field test indicated that arsenical-resistant cocklebur may exist in Missouri.

Propanil-resistant barnyardgrass. In the mid-South the most common rice weed is barnyardgrass, and the main herbicide for its control is propanil (Stam, others). In the late 1980s, growers in northeast Arkansas began reporting poor barnyardgrass control. In this area some fields had been in rice for upwards of 20 consecutive years. When barnyardgrass from these fields was tested, it tolerated as much as 20 times the normal use rate of propanil. A preliminary survey of farmers determined that a simple crop rotation greatly reduced the probability of resistance.

Technical tidbit: It often is thought that weeds change or mutate to become resistant. Biologists, however, believe that weeds do not change at all. Instead, populations change. The resistant weeds always have been present in low populations. When a particular herbicide is used, it controls the normal-susceptible types. This makes room for the population of the resistant weeds to increase. Consequently, when growers say that their "weeds have become resistant," they really mean that the population of their resistant weeds has increased greatly and the population of their susceptible weeds has decreased.

Dinitroaniline- (or DNA)-resistant goosegrass. Goosegrass is a common summer annual grass and is ranked among the 10 worst weeds in the United States. In the early 1980s, resistance was suspected in a South Carolina cotton field that had received Treflan applications for more than 10 consecutive years. Subsequent studies determined that the goosegrass was completely resistant to Balan, Basalin, Prowl, Sonolan, Surflan and Pursuit. These herbicides are all in the dinitroaniline family of chemistry (or "DNAs"). It is now believed that DNA-resistant goosegrass can be found throughout the mid-South.

Triazine-resistant weeds. A number of triazine-resistant weeds have been found across the United States. The first discovery was reported in early 1970 with groundsel. Within a few years, triazine resistance was reported with pigweed and lambsquarters. Now more than 50 species of triazine-resistant weeds exist. A triazine-resistant waterhemp (a member of the pigweed family) was discovered in Missouri in 1994.

Cross resistance

More than 100 different herbicides are on the market today. But many of these work in exactly the

same way or, in other words, have the same mode of action. Fewer than 20 plant-growth mechanisms are affected by current herbicides. ALS-herbicide resistance is a good example of the problem of cross resistance. ALS herbicides exist for many crops. The Missouri-ALS-resistant cocklebur came from a field receiving only Scepter, but the cocklebur is cross resistant to Beacon, Broadstrike and Classic. It is even resistant to a number of experimental herbicides that have yet to be labeled.

How to manage herbicide resistance

Management of resistance, once it occurs, is simple: The grower must switch to an alternative method of control (be it an alternative herbicide or a cultural control method, such as cultivation). Some private-industry personnel have recommended that the grower continue to use the same herbicide in combination with another herbicide with a different mode of action (for example, Scepter followed by Basagran where resistant cocklebur exist). Their reasoning is that the Scepter will provide control of many other weeds and that Basagran will control cocklebur. Although little data supports it, weed scientists urge growers with resistance problems to avoid particular herbicides if they have resistant weeds and to switch to herbicides with a different action mechanism. In theory, this allows the weed populations some chance to shift back to predominantly susceptible types.

Although resistance management appears simple, it is better to prevent resistance from becoming a problem. Herbicide cross resistance renders many herbicides useless at once. With the example of the Scepter-resistant Missouri cocklebur, the use of Accent, Beacon, Canopy, Classic, Permit, Pursuit or Staple could be affected. And no one can predict if or when these fields will change back to an ALS-susceptible population.

If a field is infested with herbicide-cross-resistant weeds, the farmer may lose yield because a competitive weed isn't controlled. Growers also may have higher costs if they lose the use of several economical herbicides. The use of future herbicides may even be affected. As an example, the Missouri cocklebur was resistant to Broadstrike before it was available and is already resistant to a useful cotton herbicide that, at the time this guide was written, was still not registered for use.

If you suspect a resistance problem:

1. Use herbicides with a different mechanism of action to control the escaped weed.
2. Do not let weeds go to seed.
3. Use cultural practices such as cultivation.
4. Contact your extension agent, your dealer and your sales representative.

How to prevent herbicide resistance

Table 1 lists factors that promote resistance. Avoiding these factors is a good start to preventing resistance. **Although Table 1 lists ALS herbicides as being prone to resistance, farmers still should use them when appropriate. However, they should avoid using ALS herbicides repeatedly without rotation.**

Table 2 can be used to cross-reference herbicides for their mechanism of action. Use this list to determine if you are using a resistance-prone herbicide program; that is, using **different** herbicides with the **same** mode of action. Remember, this is primarily a problem when the herbicides target the same weed species. It's fine to use an ALS-inhibiting herbicide, such as Scepter, for cocklebur in Treflan-treated soybeans and then use another ALS-inhibiting herbicide, such as Accent, for johnsongrass and shattercane control in atrazine-treated corn. The cocklebur are controlled by an ALS herbicide (Scepter) one year and by a photosynthetic inhibitor (atrazine) the next year. Grasses are being controlled by a mitotic inhibitor (Treflan) one year and an ALS-inhibiting herbicide (Accent) the other year.

However, avoid using a program such as Pursuit (an ALS-inhibiting herbicide) for shattercane control in soybeans followed by Accent or Beacon (also ALS-inhibiting herbicides) for shattercane in corn.

Other recommendations for preventing resistance include the following practices:

Cultivation. There are no known cases of cultivator-resistant weeds. No-till farmers must exercise extra care in herbicide rotation because they rely more on herbicides and less on cultivation.

Tank mixes, package mixes and sequential applications. Combinations of herbicides with different mechanisms of action, which control on the same species, also help prevent resistance. However, this strategy may only delay herbicide resistance rather than prevent it. In addition, the selection of the wrong combinations (sequentials or mixtures) of herbicides (where only one of the herbicides works on a particular weed) can cause resistant populations to build up just as rapidly as overusing a single herbicide.

Technical tidbit: More often than not, a lack of crop and herbicide rotation causes the species of weeds to change rather than the buildup of resistant varieties of weeds. For example, if you use nothing but Treflan for several years in a row, cocklebur is more likely to increase than is Treflan-resistant goosegrass. But, if you think about it, it is actually the same mechanism occurring — selection pressure causes susceptible types to decline while resistant types increase. The only difference is that the species of weeds shift in the typical case versus different types of the same weed species shifting in resistance cases.

Table 1. When and why is herbicide resistance most likely?

Factor	Explanation of effect
Lack of rotation.	The same herbicide or environment year after year results in continual selection pressure, which kills susceptible populations while allowing resistant populations to grow.
A weed that is hypersensitive to a particular herbicide.	The selection pressure is high. Very few of the susceptible types survive. Resistant types thrive and have no competition from susceptible types.
Herbicides with long residual periods.	Again, high-selection pressure, continual control of susceptible types and continual release of resistant types.
Newer herbicide with a highly specific mode of action.	The herbicide affects only one specific site in the plant; consequently, a resistant plant has to be different only in that one specific site. Older herbicides usually affect several sites, and thus, it is more unlikely for a plant to have resistance at all of the sites of action.
ALS-inhibiting herbicide.	ALS-inhibiting herbicides often fit the previous four qualifications, and, in addition, resistant types of plants apparently aren't rare. A recurring tendency exists for ALS-resistant weeds to be discovered only four to five years after the introduction of an ALS-inhibiting herbicide.
Any herbicide or weed.	Herbicide resistance does not necessarily occur when and where we expect it. It was thought that the extensive use of Accent, Beacon and Pursuit on shattercane would likely result in ALS-resistant shattercane. But a project specifically designed to investigate the probability of shattercane resistance instead discovered an ALS-resistant common waterhemp. This indicates that the overuse of any herbicide or mode of action could result in resistant weeds.

Avoiding repeat or sequential applications of the same herbicide (or active ingredient). For example, Squadron followed by Scepter or Canopy followed by Classic results in two applications of the same active ingredient in one year.

Identifying herbicides with the same mode of action

Because of the great variety of trade names and package mixes of herbicides, it is difficult for growers to keep track of which modes of action they use. Some identical active ingredients actually have multiple product names. Some package mixes deter resistance while others do not. Use Table 2 to identify herbicides that have the same mechanism of action. However, some traits may be useful for identification of a herbicide's mechanism of action without having to refer to a large table.

ALS-inhibiting herbicides include many of those used at extremely low (ounce per acre) rates. This trait correctly identifies herbicides such as Accent, Beacon, Classic, Pursuit and Scepter. The "ounce per acre" feature is lost with package mixes such as Squadron, TriScept and Broadstrike plus Dual. Another trait of ALS-inhibiting herbicides is that they act relatively slowly, stunting weeds first followed by a gradual death of the weeds.

Postemergence grass herbicides (Assure, Bugle, Fusilade, Option, Poast, Select and Whip) have the same mechanism of action. They inhibit the ACCase enzyme. At this time, these are the only herbicides that provide postemergence grass control with no broadleaf activity. Identifying a herbicide from this group is not difficult.

Photosynthetic inhibitors (atrazine, Bladex, Cotoran, Lexone, Lorox, Sencor and others) include triazine, urea and uracil types of herbicide chemistry as well as some miscellaneous herbicides, such as Basagran and Stam. With some notable exceptions, these tend to be preemergence herbicides that control broadleaf weeds. They allow weeds to germinate and emerge, but a fairly rapid burn starts at the edge of the leaves and moves inward. Large-seeded weeds may continue to grow as the "burn" follows the growing point up the plant. All photosynthetic inhibitors do not affect the same site of action in the plant; consequently, complete cross resistance is not likely.

Dinitroanilines (Prowl, Treflan and others) are almost always yellow or orange. They usually are (but not always) pre-plant incorporated herbicides that control annual grasses and small-seeded broadleaves.

Diphenyl ethers (Blazer, Cobra, Goal and Reflex) are almost exclusively postemergence soybean herbicides. They cause a rapid bronze-colored burn on weeds and soybeans, but soybeans recover.

Technical tidbit: Why are crops resistant? Why are weeds resistant? Most herbicides are selective. They control weeds but have little effect on the crop. The usual reason that a crop tolerates herbicides is that it "digests" or metabolizes the herbicide into nonpoisonous compounds before they can kill the plant. Weeds cannot metabolize the herbicide, so the herbicide remains in its original form and kills the weeds. Although it makes sense that resistant weeds are metabolizing and deactivating the herbicide, what happens in most cases is that the actual site of action within the weed is "immune" to the herbicide. For example, with the ALS-resistant cocklebur in Missouri, it is possible to apply a high-enough rate of Classic to injure soybeans, although it will have no effect on the cocklebur. This is because the high rate overloads the soybeans' ability to metabolize the herbicide, and there is enough unmetabolized herbicide to affect the ALS enzyme. In the cocklebur, none of the Classic is metabolized, but the ALS enzyme is "immune" to the Classic.

Exceptions to this rule exist. Some resistant weeds can metabolize the herbicide to nontoxic chemicals instead of having a modified binding site. Sometimes the crop has a modified, "immune" herbicide binding site. A unique type of cross resistance has been found in Australia, where certain ryegrass types have the ability to metabolize and deactivate a wide variety of herbicides. This is the worst case of cross resistance because rotating to a different mechanism of action may have no effect.

Conclusion

Herbicide resistance is a complicated subject. Many weed scientists warn of hidden dangers in rotating modern herbicides. Because so many modern herbicides have the same mechanism of action, a grower could rotate crops and herbicides but still wind up with a resistance problem. The mode of action may not change even when crops and chemicals are rotated. **But so far, the resistance cases in Missouri have come about from the same crop and same herbicide being used year after year.** Growers should keep the hidden danger of modern herbicides in the back of their minds, but the major problem in Missouri has been the violation of one of the simplest rules of good farming — rotation.

Table 2. Herbicide products, active ingredients, chemical family and mechanism of action.

Product	Active ingredient	Chemical family	Mechanism of action
AAtrex (others)	atrazine	triazine	photosynthetic inhibitor
Accent	nicosulfuron	sulfonyl-urea	ALS inhibitor
Arrosolo	molinate (Ordram) propanil (Stam)	thiocarbamate chloroacetamide	growth inhibitor photosynthetic inhibitor
Assure	quizalofop	aryl-oxy-phenoxy	ACCase inhibitor
Banvel	dicamba	benzoic	hormone initiator
Basagran	bentazon	no family	photosynthetic inhibitor
Battalion	halosulfuron	sulfonyl-urea	ALS inhibitor
Beacon	primisulfuron	sulfonyl-urea	ALS inhibitor
Benefit	dimethenamid (Frontier) pendimethalin (Prowl)	chloroacetamide dinitroaniline	growth inhibitor mitotic inhibitor
Bicep	atrazine metolachlor (Dual)	triazine chloroacetamide	photosynthetic inhibitor growth inhibitor
Bladex	cyanazine	triazine	photosynthetic inhibitor
Blazer	aciflourfen	diphenyl ether	PPO membrane disrupter
Bolero	thiobencarb	thiocarbamate	growth inhibitor
Broadstrike (several package mixes)	flumetsulam	similar to sulfonyl-urea	ALS inhibitor
Bronco	glyphosate (Roundup) alachlor (Lasso)	amino acid analog chloroacetamide	EPSP synthase inhibitor growth inhibitor
Bronate	bromoxynil (Buctril)	nitrile	photosynthetic inhibitor
Buctril	bromoxynil	nitrile	photosynthetic inhibitor
Bugle	fenoxaprop	aryl-oxy-phenoxy	ACCase inhibitor
Bullet (or Lariat)	atrazine alachlor (Lasso)	triazine chloroacetamide	photosynthetic inhibitor growth inhibitor
Butyrac	2,4-DB	phenoxy	hormone imitator
Canopy (or Preview)	chlorimuron (Classic) metribuzin (Lexone, Sencor)	sulfonyl-urea triazine	ALS-inhibitor photosynthetic inhibitor
Caparol	prometryn	no family	photsynthetic inhibitor
Clarity (or Banvel)	dicamba	benzoic	hormone imitator
Classic	chlorimuron	sulfonyl-urea	ALS inhibitor
Conclude B(Storm)	bentazon (Basagran) aciflourfen (Blazer)	no family diphenyl ether	photosynthetic inhibitor PPO membrane disrupter
Conclude G (Poast)	sethoxydim	cyclohexendione	ACCase inhibitor
Cobra	lactofen	diphenyl ether	PPO membrane disrupter
Command	clomazone	no family	carotenoid-inhibiting bleacher
Commence	clomazone (Command) trifluralin (Treflan)	no family dinitroaniline	carotenoid-inhibiting bleacher mitotic inhibitor
Concert (or Synchrony)	chlorimuron (Classic) thifensulfuron (Pinnacle)	sulfonyl-urea sulfonyl-urea	ALS inhibitor ALS inhibitor
Contour	imazethapyr (Pursuit) atrazine	imidazolinone triazine	ALS inhibitor photosynthetic inhibitor
Cotoran	fluometuron	urea	photosynthetic inhibitor
Cotton Pro	prometryn	no family	photosynthetic inhibitor
Detail	dimethenamid (Frontier) imazaquin (Scepter)	chloroacetamide imidazolinone	growth inhibitor ALS inhibitor
Direx (or Karmex)	diuron	urea	photosynthetic inhibitor
Dual	metolachlor	chloroacetamide	growth inhibitor
Eradicane/Eptam	EPTC	thiocarbamate	growth inhibitor
Exceed	prosulfuron (Peak) primisulfuron (Beacon)	sulfonyl-urea sulfonyl-ure	ALS inhibitor ALS inhibitor
Extrazine	atrazine cyanazine (Bladex)	triazine triazine	photosynthetic inhibitor photosynthetic inhibitor
Facet	quinclorac	no family	hormone imitator
Flexstar (or Reflex)	fomesafen	diphenyl ether	PPO membrane disrupter

Table 2. Herbicide products, active ingredients, chemical family and mechanism of action. *(continued)*

Product	Active ingredient	Chemical family	Mechanism of action
Freedom	alachlor (Lasso) trifluralin (Treflan)	chloroacetamide dinitroaniline	growth inhibitor mitotic inhibitor
Frontier	dimethenamid	chloroacetamide	growth inhibitor
Fusilade	fluazifop	aryl-oxy-phenoxy	ACCase inhibitor
Fusion	fluazifop (Fusilade) fenoxaprop (Whip, Bugle)	aryl-oxy-phenoxy aryl-oxy-phenoxy	ACCase inhibitor ACCase inhibitor
Galaxy	bentazon (Basagran) aciflourfen (Blazer)	no family diphenyl ether	photosynthetic inhibitor PPO membrane disrupter
Gemini	linuron (Lorox) chlorimuron (Classic)	urea sulfonyl-urea	photosynthetic inhibitor ALS inhibitor
Genate	butylate	thiocarbamate	growth inhibitor
Goal	oxyflourfen	diphenyl ether	PPO membrane disrupter
Gramoxone Extra	paraquat	bipyridilium	Ps membrane disrupter
Guardsman	atrazine dimethenamid (Frontier)	triazine chloroacetamide	photosynthetic inhibitor growth inhibitor
Harmony extra	thifensulfuron (Harmony) tribenuron (Express)	sulfonyl-urea sulfonyl-urea	ALS inhibitor ALS inhibitor
Harness (or Surpass)	acetochlor	chloroacetamide	growth inhibitor
Karmex	diuron	urea	photosynthetic inhibitor
Laddok	bentazon (Basagran) atrazine	no family triazine	photosynthetic inhibitor photosynthetic inhibitor
Landmaster	glyphosate (Roundup) 2,4-D	amino acid phenoxy	EPSP inhibitor hormone imitator
Lasso	alachlor	chloroacetamide	growth inhibitor
Lariat	alachlor (Lasso) atrazine	chloroacetamide triazine	growth inhibitor photosynthetic inhibitor
Lexone	metribuzin (same as Sencor)	triazine	photosynthetic inhibitor
Linex	linuron (Lorox)	urea	photosynthetic inhibitor
Londax	bensulfuron	sulfonyl-urea	ALS inhibitor
Lorox	linuron	urea	photosynthetic inhibitor
Lorox plus	linuron chlorimuron (Classic)	urea sulfonyl-urea	photosynthetic inhibitor ALS inhibitor
Marksman	dicamba (Banvel) atrazine	benzoic acid triazine	hormone imitator photosynthetic inhibitor
MCPA (many brands)	MCPA	phenoxy	hormone imitator
Meturon	fluometuron (Cotoran)	urea	photosynthetic inhibitor
Micro-Tech	alachlor (Lasso)	chloroacetamide	growth inhibitor
Option II	fenoxaprop (Whip, Bugle)	aryl-oxy-phenoxy	ACCase inhibitor
Ordram	molinate	thiocarbamate	growth inhibitor
Passport	imazethapyr (Pursuit) trifluralin (Treflan)	imidazolinone dinitroaniline	ALS inhibitor mitotic inhibitor
Peak	prosulfuron	sulfonyl-urea	ALS inhibitor
Pentagon (or Prowl)	pendimethalin	dinitroaniline	mitotic inhibitor
Permit	halosulfuron	sulfonyl-urea	ALS inhibitor
Pinnacle	thifensulfuron	sulfonyl-urea	ALS inhibitor
Poast	sethoxydim	cyclohexendione	ACCase inhibitor
Preview	metribuzin (Lexone, Sencor) chlorimuron	triazine sulfonyl-urea	photosynthetic inhibitor ALS inhibitor
Princep	simazine	triazine	photosynthetic inhibitor
Prowl	pendimethalin	dinitroaniline	mitotic inhibitor
Pursuit	imazethapyr	imidazolinone	ALS inhibitor
Pursuit Plus	imazethapyr (Pursuit) pendimethalin (Prowl)	imidazolinone dinitroaniline	ALS inhibitor mitotic inhibitor

Table 2. Herbicide products, active ingredients, chemical family and mechanism of action. *(continued)*

Product	Active ingredient	Chemical family	Mechanism of action
Ramrod	propachlor	chloroacetamide	growth inhibitor
Ramrod Atrazine	propachlor (Ramrod) atrazine	chloroacetamide triazine	growth inhibitor photosynthetic inhibitor
Reflex	fomesafen	diphenyl ether	PPO membrane disrupter
Rescue	naptalam (Alanap) 2,4-DB (Butyrac)	chloroacetamides phenoxy	growth inhibitor hormone imitator
Resolve	imazethapyr (Pursuit) dicamba (Banvel)	imidazolinone benzoic	ALS inhibitor hormone imitator
Resource	flumiclorac	no family	PPO membrane disrupter
Rezult B (or Basagran)	bentazon	no family	photosynthetic inhibitor
Rezult G (or Poast Plus)	sethoxydim	cyclohexendiones	ACCase inhibitor
Roundup	glyphosate	amino acid	EPSP inhibitor
Salute	metribuzin (Lexone, Sencor) trifluralin (Treflan)	triazine dinitroaniline	photosynthetic inhibitor mitotic inhibitor
Scepter	imazaquin	imidazolinone	ALS inhibitor
Scepter OT	imazaquin (Scepter) acifluorfen (Blazer)	imidazolinone diphenyl ether	ALS inhibitor PPO membrane disrupter
Select	clethodim	cyclohexendione	ACCase inhibitor
Sencor	metribuzin (same as Lexone)	triazine	photosynthetic inhibitor
Sonolan	ethalfluralin	dinitroaniline	mitotic inhibitor
Squadron	imazaquin (Scepter) pendimethalin (Prowl)	imidazolinone dinitroaniline	ALS inhibitor mitotic inhibitor
Stam	propanil	chloroacetamide	photosynthetic inhibitor
Staple	pyrithiobac	similar to sulfonyl-urea	ALS inhibitor
Storm	bentazon (Basagran) aciflourfen (Blazer)	no family diphenyl ether	photosynthetic inhibitor PPO membrane disrupter
Surpass (or Harness)	acetochlor	chloroacetamide	growth inhibitor
Surpass 100	atrazine acetochlor (Harness)	triazine chloroacetamide	photosynthetic inhibitor growth inhibitor
Sutan	butylate	thiocarbamate	growth inhibitor
Sutazine	butylate (Sutan) atrazine	thiocarbamate triazine	growth inhibitor photosynthetic inhibitor
Synchrony	chlorimuron (Classic) thifensulfuron (Pinnacle)	sulfonyl-urea sulfonyl-urea	ALS inhibitor ALS inhibitor
Topnotch (or Surpass)	acetochlor	chloroacetamide	growth inhibitor
Tornado/Typhoon	fluazifop (Fusilade) fomesafen (Reflex)	aryl-oxy-phenoxy diphenyl ether	ACCase inhibitor PPO membrane disrupter
Tough	pyridate	no family	photosynthetic inhibitor
Treflan	trifluralin	dinitroaniline	mitotic inhibitor
Trific	trifluralin (Treflan)	dinitroaniline	mitotic inhibitor
Trilin	trifluralin (Treflan)	dinitroaniline	mitotic inhibitor
TriScept	imazaquin (Scepter)	imidazolinone	ALS inhibitor
Turbo	metribuzin (Lexone, Sencor) metolachlor (Dual)	triazine chloracetamide	photosynthetic inhibitor growth inhibitor
Vernam	vernolate	thiocarbamate	growth inhibitor
Whip	fenoxaprop (Bugle)	aryl-oxy-phenoxy	ACCase inhibitor
Zorial	norflurzon	no family	carotenoid-inhibiting bleacher
2,4-D	2,4-D	phenoxy	hormone imitator

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