Herbicides kill plants by disrupting an essential physiological process. This normally is accomplished by the herbicide specifically binding to a single protein. The target protein is referred to as the herbicide “site of action.” Herbicides in the same family generally have the same site of action. The mechanism by which a herbicide kills a plant is known as its “mode of action.” For example, triazine herbicides interfere with photosynthesis by binding to the D1 protein involved in photosynthetic electron transfer. Thus, the site of action for triazines is the D1 protein, whereas the mode of action is the disruption of photosynthesis. An understanding of herbicide mode of action is essential for diagnosing crop injury or off-target injury problems and for designing weed management programs with a low risk of selecting for herbicide-resistant weed populations.

ACCase Inhibitors
The ACCase enzyme is involved in the synthesis of fatty acids. Two herbicide families attack this enzyme. Aryloxyphenoxypropionate (commonly referred to as “fops”) and cyclohexanedione (referred to as “dms”) herbicides are used postemergence, although some have limited soil activity (e.g. fluazifop). ACCase inhibitors are active only on grasses, and selectivity is due to differences in sensitivity at the site of action, rather than differences in absorption or metabolism of the herbicide. Most herbicides in this class are translocated within the phloem of grasses. The growing points of grasses are killed and rot within the stem.

Herbicides listed in italics in this section are tank-mix components representing a different chemical family.

At sublethal rates, irregular bleaching of leaves or bands of chlorotic tissue may appear on affected leaves. Resistant weed biotypes have evolved following repeated applications of these herbicides. An altered target site of action is responsible for the resistance. Mode of action: ACCase.

Product  Active ingredients
Aryloxyphenoxypropionate
Assure II _______ quizalofop-p-ethyl
Fusilade DX ____ fluazifop-p-buty1
Fusion _________ fluazifop-p-buty1 + fenoxaprop
Hoelon* _______ diclofop

Cyclohexanedione
Poast, Poast Plus _ sethoxydim
Select ___________ clethodim

ALS Inhibitors
Several chemical families interfere with acetolactate synthase (ALS), an enzyme involved in the synthesis of branched-chain amino acids, specifically valine, leucine, and isoleucine. These amino acids are necessary for protein synthesis and plant growth. Generally, these herbicides are absorbed in plant roots and foliage and are readily translocated in the xylem and phloem. The herbicides accumulate in meristematic regions of the plant and the herbicidal effects are first noted there. Symptoms include plant stunting, chlorosis (yellowing), and tissue necrosis (death) and are evident 1 to 4 weeks after herbicide application, depending upon the plant species and environmental conditions. Soybeans and other affected broad-leaves often develop reddish veins on undersides of leaves.

Symptoms in corn include reduced secondary root formation, stunted roots, shortened internodes, leaf malformations (chlorosis, window-paning) and nutrient deficiency. However, symptoms typically are not distinct or consistent. Factors such as soil moisture, temperature, and soil compaction can enhance the occurrence of injury or may mimic the herbicide injury. Some ALS inhibiting herbicides have long soil residual properties and may carry over and injure sensitive rotational crops. Herbicide resistant weed biotypes possessing an altered site of action have evolved after repeated applications of these herbicides. Mode of action: ALS.

Product  Active ingredients
Imidazolinones
Lightning _______ imazethapyr + imazapyr
Pursuit ________ imazethapyr
Pursuit Plus______ imazethapyr + pethoximethalin
Raptor _________ imazamox
Scepter __________ imazquin
Squadron_________ imazaquin + pethoximethalin

Sulfonanilides
FirstRate/Amplycloransulam-methyl
Hornet WDG ___ flumetsulam + clopyralid
Python _________ flumetsulam

Sulfonylureas
Accent __________ nicosulfuron
Accent Gold ______ nicosulfuron + rimsulfuron + clopyralid + flumetsulam
Ally/Cimarron ___ metsulfuron

* Restricted use pesticides
**Basis** __________ rimsulfuron + thifensulfuron methyl
**Basis Gold*** _____ rimsulfuron + nicosulfuron + atrazine
**Beacon_________** primisulfuron
**Canopy _______** chlorimuron-ethyl + metribuzin
**Canopy XL _____** chlorimuron + sulfentrazone
**Celebrity Plus ___** nicosulfuron + dicamba + diflufenzopyr
**Classic_________** chlorimuron-ethyl
**Equip __________** foramsulfuron + iodosulfuron + safener
**Exceed, Spirit ___** prosulfuron + primisulfuron
**Express_________** tribenuron
**Harmony GT_______** thifensulfuron methyl
**NorthStar _______** primisulfuron + dicamba
**Option___________** foramsulfuron + safener
**Permit___________** halosulfuron
**Steadfast _______** nicosulfuron + Rimsulfuron + atrazine
**Synchrony STS __** chlorimuron-ethyl + thifensulfuron methyl
**Steadfast ATZ ___** nicosulfuron + Rimsulfuron + atrazine
**Yukon___________** halosulfuron methyl + dicamba

**Microtubule Inhibitors**
Dinitroaniline (DNA) herbicides inhibit cell division by interfering with the formation of microtubules. Dinitroaniline herbicides are soil-applied and absorbed mainly by roots. Very little herbicide translocation into plants occurs, thus the primary herbicidal effect is on root development. Soybean injury from DNA herbicides is characterized by root pruning. Roots that do develop are thick and short. Hypocotyl swelling also occurs. The inhibited root growth causes tops of plants to be stunted and often to demonstrate a dark green color. Corn injured by DNA carryover demonstrates root pruning and short, thick roots. Leaf margins may have a reddish color. Since DNAs are subject to little movement in the soil, such injury is often spotty due to localized concentrations of the herbicide. Early season stunting from DNA herbicides typically does not result in significant yield reductions. Mode of action: microtubule.

**Synthetic Auxins**
Several chemical families cause abnormal root and shoot growth by upsetting the plant hormone (auxin) balance. These herbicides are primarily effective on broadleaf species. Uptake can occur through seeds or roots with soil-applied treatments or leaves when applied postemergence. Synthetic auxins translocate throughout plants and accumulate in areas of high growth activity. Corn injury may occur in the form of onion leafing, proliferation of roots, or abnormal brace root formation. Corn stalks may become brittle following application; this response usually lasts for 7 to 10 days following application. The potential for injury increases when applications are made to corn larger than 10 to 12 inches in height. Soybean injury from synthetic auxin herbicides is characterized by cupping and crinkling of leaves. Soybeans are extremely sensitive to dicamba; however, early season injury resulting only in leaf malformation usually does not affect yield potential. Soybeans occasionally develop symptoms characteristic of dicamba in the absence of this herbicide. This response is poorly understood, but usually develops during periods of rapid growth or following stress from other postemergence herbicide applications. Dicamba has a high vapor pressure and may move off target due to volatilization. Mode of action: auxin.
**Photosystem II Inhibitors**

Several families of herbicide bind to a protein involved in electron transfer in Photosystem II (PSII). These herbicides inhibit photosynthesis, which may result in chlorosis of plant leaves followed by necrosis of leaf tissue. A secondary substance formed as a result of photosynthesis inhibition may be responsible for plant death. When PSII inhibitors are applied to the leaves, uptake occurs into the leaf but very little movement out of the leaf occurs. Injury to corn occurs as yellowing of leaf margins and tips followed by browning, whereas injury to soybean occurs as yellowing or burning of outer leaf margins. The entire leaf may turn yellow, but veins usually remain somewhat green (interveinal chlorosis). Lower leaves are most affected, and new leaves may be unaffected. Triazine and urea herbicides generally are absorbed both by roots and foliage, whereas benzothiadiazole and nitrile herbicides are absorbed primarily by plant foliage. Triazine-resistant biotypes of several weed species have been confirmed in Iowa following repeated use of triazine herbicides. Although the other PSII herbicides attack the same target site, they bind on a different part of the protein and remain effective against triazine resistant weeds. Mode of action: PSII.

<table>
<thead>
<tr>
<th>Product</th>
<th>Active ingredients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzothiadiazoles</td>
<td></td>
</tr>
<tr>
<td>Basagran</td>
<td>bentazon</td>
</tr>
<tr>
<td>Galaxy, Storm</td>
<td>bentazon + acifluorfen</td>
</tr>
<tr>
<td>Laddok*</td>
<td>bentazon + atrazine</td>
</tr>
<tr>
<td>Nitriles</td>
<td></td>
</tr>
<tr>
<td>Bison</td>
<td>bromoxynil + MCPA</td>
</tr>
<tr>
<td>Buctril</td>
<td>bromoxynil</td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Buctril Gel</td>
<td>bromoxynil</td>
</tr>
<tr>
<td>Buctril + Atrazine*</td>
<td>bromoxynil + atrazine</td>
</tr>
<tr>
<td>Connect</td>
<td>bromoxynil</td>
</tr>
<tr>
<td>Moxy</td>
<td>bromoxynil</td>
</tr>
<tr>
<td>Moxy + Atrazine*</td>
<td>bromoxynil + atrazine</td>
</tr>
</tbody>
</table>

**Photosystem I Inhibitors**

Herbicides in the bipyridilium family rapidly disrupt cell membranes, resulting in wilting and tissue death. They capture electrons moving through Photosystem I (PSI) and produce highly destructive compounds. Very little translocation of bipyridilium herbicides occurs due to loss of plant membrane structure. Injury occurs only where the herbicide spray contacts the plant. Complete spray coverage is essential for weed control. The herbicide molecules carry strong positive charges that cause them to be very tightly adsorbed by soil colloids. Consequently, bipyridilium herbicides have no significant soil activity. Injury to crop plants from paraquat drift occurs in the form of spots of dead leaf tissue wherever spray droplets contact the leaves. Typically, slight drift injury to corn, soybeans, or ornamentals from a bipyridilium herbicide does not result in significant growth inhibition. Mode of action: PSI.

<table>
<thead>
<tr>
<th>Product</th>
<th>Active ingredients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aryl triazolinones</td>
<td></td>
</tr>
<tr>
<td>Aim</td>
<td>carfentrazone-ethyl</td>
</tr>
<tr>
<td>Authority/Spartan</td>
<td>sulfentrazone + cloransulam-methyl</td>
</tr>
<tr>
<td>Gauntlet</td>
<td>sulfentrazone + chlorimuron</td>
</tr>
<tr>
<td>Canopy XL</td>
<td>sulfentrazone + clomazone</td>
</tr>
<tr>
<td>Command Xtra</td>
<td>sulfentrazone + clomazone</td>
</tr>
</tbody>
</table>

**Protoporphyrinogen Oxidase (PPO) Inhibitors**

The specific site of action is an enzyme involved in synthesis of a precursor of chlorophyll; the enzyme is referred to as PPO. Postemergence applied diphenyl ether herbicides (e.g. acifluorfen) kill weed seedlings through contact action, membrane destruction, and photosynthesis inhibition. Thorough plant coverage by the herbicide spray is required. Applying the herbicide prior to prolonged cool periods or during hot, humid conditions will result in crop injury. Injury symptoms range from speckling of foliage to necrosis of whole leaves. Under extreme situations, herbicide injury has resulted in the death of the terminal growing point, which produces short, bushy soybean plants. Most injury attributable to diphenyl ether herbicides is cosmetic and does not affect yields. The arylo triazolinones herbicides are absorbed both by roots and foliage. Susceptible plants emerging from soils treated with these herbicides turn necrotic and die shortly after exposure to light. Soybeans are most susceptible to injury if heavy rains occur when beans are cracking the soil surface. Mode of action: PPO.
Diphenyl Ethers
Blazer, UltraBlazer, acifluorfen
Cobra, lactofen
Flexstar, Reflex, fomesafen
Goal, oxyfluorfen
Phenylphthalimides
Resource, flumiclorac
Valor, flumioxazin

Enolpyruvyl Shikimate Phosphate Synthase (EPSPS) Inhibitors
Glyphosate is a substituted amino acid that interferes with amino acid synthesis by inhibiting the EPSPS enzyme. This enzyme is involved in the synthesis of several essential amino acids. Glyphosate is nonselective and is very tightly bound in soil so no root uptake occurs. Applications must be made to plant foliage. Translocation occurs out of leaves to all plant parts including underground storage organs of perennial weeds. Translocation is greatest when plants are actively growing. Injury symptoms are fairly slow in appearing. Leaves slowly wilt, turn brown, and die. Sub-lethal rates of glyphosate sometimes produce phenoxy-type symptoms with feathering of leaves (parallel veins) or proliferation of vegetative buds. Mode of action: EPSPS.

Product Active ingredients
Roundup Ultra/
Roundup UltraMax/
Roundup UltraDry/
Roundup WEATHERMAX/
Glyphomax/Glyphomax Plus/
Rodeo/Touchdown/
others glyphosate
ReadyMaster ATZ* glyphosate + atrazine
Extreme glyphosate + imazethapyr

Glutamine Synthetase Inhibitors
Glufosinate (Liberty) inhibits the enzyme glutamine synthetase, causing a buildup of ammonia in the plant which becomes phytotoxic. Glufosinate is relatively fast acting and provides effective weed control in three to seven days. Symptoms appear as chlorotic lesions on the foliage followed by necrosis. There is limited translocation of glufosinate within plants. The herbicide has no soil activity. Liberty is nonselective except to crops that carry the Liberty-Link gene. Mode of action: GS.

Product Active ingredients
Liberty glufosinate
Liberty ATZ* glufosinate + atrazine

Hydroxyphenyl Pyruvate Dioxygenase (HPPD) Inhibitors
Isoxaflutole (Balance Pro) and mesotrione (Callisto) bind to HPPD, an enzyme involved in the synthesis of carotene pigments. Injury symptoms include bleaching or chlorosis. Although the chemicals have the same site of action, they are not chemically related. The herbicides are absorbed both by roots and foliage. Mode of action: bleacher.

Product Active ingredients
Balance* isoxaflutole
Balance Pro* isoxaflutole + flufencat
Epic* isoxaflutole + mesotrione + atrazine + S-metolachlor
Callisto mesotrione
Lumax mesotrione + atrazine + S-metolachlor

Diterpene Inhibitors
Clomazone interferes with the synthesis of the same pigments as the HPPD inhibitors, buts acts at a different enzyme within the metabolic pathway. Sensitive plants exposed to the herbicide turn white. Clomazone is xylem mobile and taken up in roots and shoots. Differential metabolism of clomazone confers tolerance to plants. Clomazone has a relatively high vapor pressure and may volatilize off the soil surface resulting in off-target injury. Mode of action: bleacher.

Product Active ingredients
Command clomazone
Command Xtra clomazone + sulfentrazone

Auxin Transport Inhibitors
Diflufenzopyr (Distinct) has a unique mode of action in that it inhibits the transport of auxin, a naturally occurring growth regulator. Diflufenzopyr is primarily active on broadleaf species, but it may suppress certain grasses under favorable conditions. Diflufenzopyr is primarily active through foliar uptake, but it can be absorbed through the soil for some residual activity. Injury symptoms are similar to growth regulator herbicides. Mode of action: auxin transport.

Product Active ingredients
Distinct diflufenzopyr + dicamba

Lipid Synthesis Inhibitors
Although the specific target site for the thiocarbamate herbicides has not been identified, it is believed the primary site of action is lipid synthesis. In grasses, thiocarbamate herbicides inhibit meristem activity and cause abnormal emergence of leaves from
the coleoptile. The growth of susceptible broadleaf weeds is inhibited, and plants exhibit cupped or crinkled leaves. Uptake may occur through seeds, shoots, and roots; shoots are more affected than roots. These herbicides are soil-applied and must be physically incorporated into the soil due to volatility characteristics. Corn injury from thiocarbamate herbicides is demonstrated by leaves not properly unrolling from the coleoptile. Leaves are stunted and twisted, often appearing knotted.

In recent years, antidotes or safeners have been developed that help to prevent thiocarbamate injury to corn. These are formulated directly with the herbicide. The protective mechanism of these antidotes is not known, but they may enable corn to more rapidly degrade the herbicides. The antidotes are formulated directly with the herbicides; Sutan+ contains R-25788, and Eradicane contains R-29148.

Soybean injury from thiocarbamate herbicides occurs as slowed emergence and crinkling of leaves on seedling plants. The antidotes or safeners do not protect soybeans from thiocarbamate herbicides. Mode of action: lipid.

Product | Active ingredients
---|---
Eradicane | EPTC +R- 29148
Eradicane Extra | EPTC +R- 29148 + R- 33865
Sutan + | butylate + R- 29148
Sutazine* | butylate + R- 29148 + atrazine

**Unknown Site of Action**

Herbicides in the amide family (also referred to as acetanilides or acetamides) inhibit root and shoot growth causing stunted, malformed seedlings. The specific site of action and mode of action of this herbicide family is unknown. Normal cell division, cell elongation, and protein synthesis are potentially inhibited. The herbicides must be present in early stages of germination and growth of weeds for effective control. These herbicides are most effective on annual grass weeds, although some small-seeded annual broadleaf weeds are also sensitive. Injury symptoms to corn from these herbicides include leafing out underground and failure of leaves to properly unfurl. Soybean injury from these herbicides occurs in the form of a shortened mid-vein in the leaflets resulting in crinkling and a heart-shaped appearance. Dimethenamid (Frontier) and flufenacet (Axiom) have slightly different chemical structures than the amide herbicides, but it is believed they kill plants in the same manner as the amides. Mode of action: unknown.

Product | Active ingredients
---|---
Axiom, Domain | flufenacet + metribuzin
Axiom AT | flufenacet + metribuzin + atrazine
Bicep II* | flufenacet + metribuzin + atrazine
MAGNUM* /Cinch | S-metolachlor + atrazine + safener
Bicep Lite II* | S-metolachlor + atrazine + safener
MAGNUM | S-metolachlor + atrazine + safener
Boundary | S-metolachlor + metribuzin
Bullet* | alachlor + atrazine
Define | flufenacet
Degree* | acetochlor + safener
Degree Xtra* | acetochlor + atrazine + safener
Dual II | S-metolachlor + atrazine + safener
Domain | flufenacet + metribuzin
Epic* | flufenacet + isoxaflutole
FieldMaster* | acetochlor + atrazine + glyphosate + safener
Frontier | dimethenamid
FullTime* | acetochlor + atrazine + safener
Guardsman | dimethenamid + atrazine
Harness* | acetochlor + MON 13900
Harness Xtra* | acetochlor + atrazine + safener
Harness Xtra 5.6L* | acetochlor + atrazine + safener
Lariat* | alachlor + atrazine
Lasso*, Intro, MicroTech* | alachlor
Outlook | dimethenamid-P
Surpass* | acetochlor + safener
Surpass 100* | acetochlor + atrazine + safener
TopNotch* | acetochlor + safener

* Restricted use pesticides

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