

# Herbicide Site of Action and Injury Symptoms

(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. 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.

## **ALS Inhibitors – 2**

Several chemical families interfere with acetolactate synthase (ALS), an enzyme involved in the synthesis of the essential branched chain amino acids (valine, leucine, and isoleucine). This enzyme is also called acetohydroxy acid synthase (AHAS). 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 observed 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 sensitive broad-leaf plants often develop reddish veins on the undersides of leaves. Symptoms in corn include reduced secondary root formation, stunted roots, shortened internodes, leaf malformations (chlorosis, window-paning) and nutrient deficiencies. 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.

## **Microtubule Inhibitors – 3**

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 in 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. 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.

## **Synthetic Auxins – 4**

Several chemical families cause abnormal root and shoot growth by upsetting the plant hormone (i.e. auxin) balance. These herbicides are primarily effective on broadleaf species, however some monocots are also sensitive. 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. 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 auxin herbicides in the absence of this herbicide. This response is poorly understood, but usually develops during periods of rapid growth, low temperatures or following stress from other postemergence herbicide applications. Dicamba has a high vapor pressure and may move off target due to volatilization.

### **Photosystem II Inhibitors – 5, 6, 7**

Several families of herbicide bind to a protein involved in electron transfer in Photosystem II (PSII). These herbicides inhibit photosynthesis, which may result in interveinal chlorosis of plant leaves followed by necrosis of leaf tissue. Other secondary substances resulting from 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 (5) and urea (7) herbicides generally are absorbed both by roots and foliage, whereas benzothiadiazole (6) and nitrile (6) 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.

### **Photosystem I Inhibitors - 22**

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 secondary plant compounds. Very little translocation of bipyridilium herbicides occurs due to loss of 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.

### **Protoporphyrinogen Oxidase (PPO) Inhibitors – 14**

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., aciflurofen) kill weed seedlings through contact action, membrane destruction, and ultimately 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 aryl triazolines 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.

### **Carotenoid synthesis inhibitors –13, 27**

Herbicides in these families inhibit the synthesis of the carotene pigments. Several different enzymes in the synthesis of carotenoids are targeted by herbicides. Clomoxone (Command) inhibits DOXP (13), whereas the other bleaching herbicides used in corn (Callisto, Balance Flexx, Laudis, Impact) inhibit HPPD (27). Carotenes are pigments with a primary function of dissipating the oxidative energy of compounds (singlet oxygen) produced during photosynthesis. In the absence of carotenes, chlorophyll and membranes are destroyed. The loss of chlorophyll results in bleaching of affected tissues. These herbicides are xylem mobile and absorbed by both roots and leaves.

### **Enolpyruvyl Shikimate Phosphate Synthase (EPSPS) Inhibitors – 9**

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) and proliferation of vegetative buds, or in some cases cause bleaching of foliage.

### **Glutamine Synthetase Inhibitors – 10**

Glufosinate (Liberty, Ignite) 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. Ignite is nonselective except to crops that carry the Liberty Link gene. **Iowa State University Extension Weed Science – [www.weeds.iastate.edu](http://www.weeds.iastate.edu) 24**

## Fatty acid and lipid synthesis inhibitors – 8

The specific site of action for the thiocarbamate herbicides (EPTC, butylate) is unknown, but it is believed they may conjugate with acetyl coenzyme A and other molecules with a sulfhydryl component. Interference with these molecules results in the disruption of fatty acid and lipid synthesis, along with other processes. Thiocarbamate herbicides are soil applied and require mechanical incorporation due to high volatility. Leaves of grasses injured by thiocarbamates do not unroll properly from the coleoptiles, resulting in twisting and knotting. Broadleaf plants develop cupped or crinkled leaves.

## Very long chain fatty acid synthesis inhibitors (VLCFA) –15

Several chemical families (acetamide, chloroacetamide, oxyacetamide and tetrazolinone) are thought to inhibit synthesis of very long chain fatty acids. VLCFA are believed to play important roles in maintaining membrane structure. These herbicides affect susceptible weeds before emergence and have little effect on emerged plants. They are most effective on annual grasses, but have activity on certain small-seeded broadleaves. Soybean injury occurs in the form of a shortened mid-vein in leaflets, resulting in crinkling and a heart-shaped appearance. Leaves of grasses, including corn, damaged by these herbicides fail to unfurl properly, and may emerge underground.

## Auxin Transport Inhibitors – 19

Diflufenzopyr (Distinct) has a unique mode of action in that it inhibits the transport of auxin, a naturally occurring plant-growth regulator. It is sold only in combination with dicamba. 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. Status (dicamba + diflufenzopyr) includes a safener to improve crop safety.

### aryloxyphenoxy-propanoate

Assure II, others	quizalofop-p-ethyl
Fusilade DX	fluzafop-p-butyl
Fusion	fluzafop-p-butyl + fenoxaprop
Hoelon	diclofop

### cyclohexanediones

Poast, Poast Plus	sethoxydim
Select, Arrow, others	clethodim

### imidazolinones

Authority Assist	imazethapyr + sulfentrazone
Lightning	imazethapyr + imazapyr
OpTill	imazethapyr + saflufenacil
Pursuit	imazethapyr
Pursuit Plus	imazethapyr + pendimethalin
Raptor	imazamox
Scepter	imazaquin
Squadron	imazaquin + pendimethalin

### sulfonanilides

FirstRate, Amplify	chloransulam
Hornet WDG	flumetsulam + clopyralid
Python	flumetsulam
SureStart/TripleFlex	flumetsulam + clopyralid + acetochlor

### sulfonylureas

Accent	nicosulfuron
Accent Q	nicosulfuron +safener
Ally, Cimarron	metsulfuron
Authority XL	chlorimuron + sulfentrazone
Basis	rimsulfuron + thifensulfuron
Beacon	primisulfuron
Canopy	chlorimuron + metribuzin
Canopy EX	chlorimuron + tribenuron
Classic	chlorimuron
Envive	flumioxazin + thifensulfuron + chlorimuron
Enlite	flumioxazin + thifensulfuron + chlorimuron
Equip	foramsulfuron + iodosulfuron + safener
Exceed, Spirit	prosulfuron + primisulfuron
Express	tribenuron

Freestyle	chlorimuron + thifensulfuron + tribenuron
Harmony GT	thifensulfuron
Instigate	chlorimuron + rimsulfuron + mesotrione
NorthStar	primisulfuron + dicamba
Option	foramsulfuron + safener
Permit, Halofax	halosulfuron
Prequel	rimsulfuron + isoxaflutole
Require Q	rimsulfuron + dicamba
Resolve Q	rimsulfuron + thifensulfuron + safener
Steadfast Q	nicosulfuron + rimsulfuron + safener
Synchrony STS	chlorimuron + thifensulfuron
Traverse	chlorimuron + rimsulfuron
Trigate	rimsulfuron + tribenuron methyl + mesotrione
Valor XLT	flumioxazin + chlorimuron
Yukon	halosulfuron + dicamba
<b>Other</b>	
Corvus	thiencarbazone-methyl + isoxaflutole safener

#### **dinitroanilines**

Balan	benefin
Commence	trifluralin + clomazone
Prowl H <sub>2</sub> O, Pentagon, Pendimax, others	pendimethalin
Sonalan	ethalfluralin
Surflan	oryzalin
Treflan, others	trifluralin

#### **benzoic**

Banvel, Clarity, others	dicamba
Distinct, Status	dicamba + diflufenzopyr
NorthStar	dicamba + primisulfuron
Require Q	rimsulfuron + dicamba
Yukon	dicamba + halosulfuron

#### **phenoxy**

many	MPCA
many	2,4-D
Butyrac, Butoxone	2,4-DB

#### **pyridines**

Crossbow	triclopyr + 2,4-D
Grazon P&D	picloram + 2,4-D
GrazonNext, ForeFront R&P	aminopyralid + 2,4-D
Hornet WDG	clopyralid + flumetsulam
PastureGard	triclopyr + fluroxypyr
Redeem	triclopyr + clopyralid
Remedy Ultra, Pathfinder II, many others	triclopyr