Herbicide Mode of Action and Injury Symptoms

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North Central Regional Extension Publications are subject to peer review and prepared as a part of the Cooperative Extension activities of the 13 land-grant universities of the 12 North Central States, in cooperation with the Extension Service—U.S. Department of Agriculture, Washington, D.C. The following states cooperated in making this publication available.

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In cooperation with NCR Educational Materials Project
Issued in furtherance of Cooperative Extension work, Acts of Congress of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture and Cooperative Extension Services of Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota and Wisconsin. Katherine Fennelly, Dean and Director of the University of Minnesota Extension Service, St. Paul, Minnesota 55108.

Herbicide Mode of Action

To be effective, herbicides must **1)** adequately contact plants; **2)** be absorbed by plants; **3)** move within the plants to the site of action, without being deactivated; and **4)** reach toxic levels at the site of action. The application method used, whether preplant incorporated, preemergence, or postemergence, determines whether the herbicide will contact germinating seedlings, roots, shoots, or leaves of plants.

The term *mode of action* refers to the sequence of events from absorption into plants to plant death. The mode of action of the herbicide influences how the herbicide is applied. For example, contact herbicides that disrupt cell membranes, such as acifluorfen (Blazer) or paraquat (Gramoxone Extra), need to be applied postemergence to leaf tissue in order to be effective. Seedling growth inhibitors, such as trifluralin (Treflan) and alachlor (Lasso), need to be applied to the soil to effectively control newly germinated seedlings.

Soil-Applied Herbicide Activity in Plants

Because the seeds of many weed species are quite small and germinate within 0.5 to 1.0 inch of the soil surface, it is important that soil-applied herbicides be positioned in the top 1 to 2 inches of soil to be effective. Herbicide positioning can be accomplished by mechanical incorporation or rainfall. Once a herbicide comes in contact with the plant, absorption through the roots or shoots is very important. A herbicide that is absorbed through the roots will be taken up as long as the herbicide-treated soil remains in contact with the absorbing region near the root tips. As the roots grow to greater soil depths, herbicide uptake declines. Therefore, weeds not killed before the root tips grow out of the herbicide-treated soil are likely to survive.

Many soil-applied herbicides are absorbed through plant shoots while they are still underground and may kill or injure the shoots before they emerge from the soil. Volatile herbicides such as the thiocarbamates (e.g., EPTC [Eradicane]) and the dinitroanilines (e.g., trifluralin [Treflan]) can penetrate the shoot as gases. Less volatile herbicides such as the acetanilides (e.g., alachlor [Lasso]) are absorbed into the shoot as liquids. Physical and environmental factors that promote rapid crop emergence reduce the length of time that a plant is in contact with a soil-applied herbicide and, therefore, reduce the possibility of crop injury.

Herbicides differ in their ability to translocate (i.e., move) within a plant. The soil-applied dinitroaniline herbicides (e.g., trifluralin [Treflan]) are not mobile within the plant. Therefore, their injury symptoms are confined to the site of uptake. Other herbicides are mobile within the plant. For example, soil-applied atrazine is absorbed by plant roots and moves upward within the water transport system of the plant (i.e., xylem) to be concentrated in the leaves. In general, injury symptoms will be most prominent at the site where the mobile herbicides concentrate.

Postemergence Herbicide Activity in Plants

Effective postemergence herbicide application is dependent upon adequate contact with above-ground plant shoots and leaves. Therefore, it is important that spray pressure and volume be adjusted for adequate plant coverage. Also, it is very important that the proper nozzles be used. Hollow-cone or flat-fan nozzles are generally recommended. Read the herbicide label for details.

For postemergence herbicides, the chemical and physical relationships between the leaf surface and the herbicide often determine the rate and amount of uptake. Factors such as plant size and age, water stress, air temperature, relative humidity, and herbicide additives can influence the rate and amount of herbicide uptake. Additives such as crop oil concentrates, surfactants, or liquid fertilizer solutions (e.g., UAN) can increase herbicide uptake by a plant. Application of herbicides under hot and dry conditions or application to older and larger weeds or weeds under water stress can decrease the amount of herbicide uptake. Differences in the rate and amount of herbicide uptake influence the potential for crop injury and weed control and often explain the year to year variation in the effectiveness of the herbicide. Also, the faster a herbicide is absorbed by a plant, the less likely it will be that rain will wash the herbicide off the plants.

Like soil-applied herbicides, postemergence herbicides differ in their ability to move within a plant. For adequate weed control, nonmobile postemergence herbicides must thoroughly cover the plant. Nonmobile herbicides are often called contact herbicides and include the bipyridylium, diphenylether, benzothiadiazole, and nitrile families. Other herbicides are mobile within the plant and can move from the site of application to their site of herbicidal activity. For example, growth regulator herbicides such as 2,4-D and dicamba (Banvel) move both upward and downward within a plant's food transport system (i.e., the phloem) to the growing points of the shoots and roots. In general, injury symptoms will be most prominent at the sites at which the mobile herbicides concentrate.

Herbicide Selectivity

Plants that can rapidly degrade or deactivate a herbicide can escape that herbicide's toxic effects. Corn is tolerant to the triazine herbicides because it quickly deactivates these herbicides by binding them to naturally occurring plant chemicals. Soybean tolerance to metribuzin (Sencor, Lexone) is at least partially due to the deactivation of the herbicide by conjugating (i.e., binding) to plant sugar molecules.

Situations may occur in which a crop may be injured by a herbicide to which it is normally tolerant. This often occurs because environmental stresses such as hot or cold temperatures, high relative humidity, or hail decrease a plant's natural ability to reduce herbicide uptake or deactivate a herbicide. Postemergence cyanazine (Bladex) injury to corn under cold and wet weather conditions is a good example of environmentally induced herbicide injury. An excessive application of herbicide, due to misapplication,

Table 1. *Herbicide classification and geographic location of weeds that have developed herbicide resistant biotypes.*

Herbicide Family	Herbicides	Weeds	Country
Dinitroaniline	Trifluralin	Goosegrass Green foxtail	USA Canada
Bipyridylium	Paraquat	Hairy Fleabane	Egypt
Arlyoxyphenoxy-propionate	Diclofop	Annual Ryegrass	Australia
Triazine	Atrazine/ Simazine	Common Groundsel, Lambsquarters, Pigweed, Kochia, Annual Bluegrass, Witchgrass, Downy Brome	USA, Canada
Sulfonylurea	Chlorsulfuron	Kochia, Russian Thistle, Prickly Lettuce	USA

can also injure a tolerant crop by overwhelming the crop's herbicide degradation and deactivation systems.

Herbicide Resistance

A number of weed species that were once susceptible and easily managed by certain herbicides have developed resistance. These weeds are no longer controlled by applications of previously effective herbicides. To date, at least 53 species of weeds are resistant to at least five different herbicide families. Some well-known herbicides and resistant species are presented in **Table 1.**

Herbicide resistance probably develops through the selection of naturally occurring biotypes of weeds exposed to a particular family of herbicides over a period of years. A biotype is a population of plants within the same species that has specific traits in common. Resistant plants survive, go to seed, and create new generations of herbicide resistant weeds.

Mechanisms for resistance vary depending on herbicide family. Resistant biotypes may have slight biochemical differences from their susceptible counterparts that eliminates sensitivity to certain herbicides. For example, in sulfonylurea susceptible plants, a herbicide attaching or binding to an enzyme (acetlactate synthase or ALS) is responsible for disrupting amino acid biosynthesis (see **Figure 1**). Sulfonylurea herbicide resistant plants have a modified ALS enzyme that prevents herbicide binding.

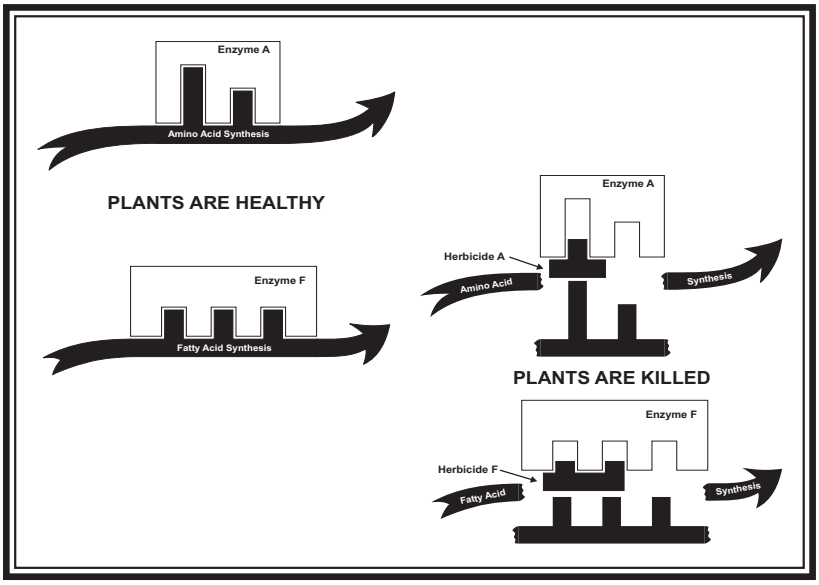

Figure 1. *Enzymes—Enzymes function as steps in biological processes. Enzymes are also extremely specialized in their function. As a result, many different enzymes are involved with the many different biological processes that occur within a plant. Some herbicides can stop specific enzymes from functioning, resulting in a disruption of specific plant processes; this often leads to the death of the plant.This herbicide-enzyme relationship is very specific and any chemical modification of the herbicide or enzyme can eliminate herbicidal activity.*

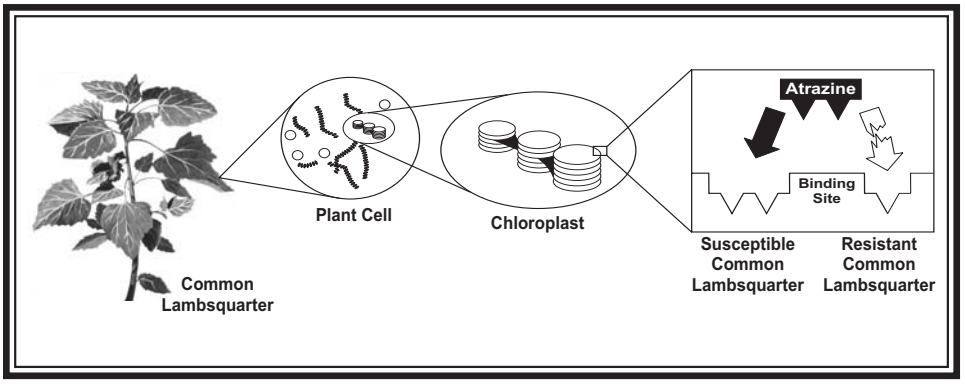

Figure 2. *Photosynthetic Inhibitors—The photosynthetic process occurs within a plant cell's chloroplasts. Certain herbicides can inhibit photosynthesis by binding to specific sites within the chloroplast. The relationship of a herbicide to the chloroplast binding site is very specific and any modification of the herbicide or binding site can eliminate herbicidal activity.*

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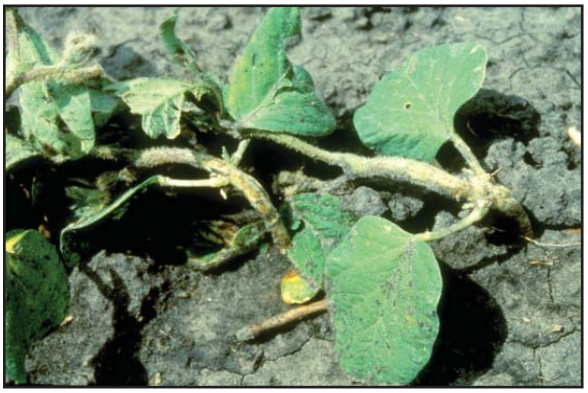

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Also, while photosynthesis is inhibited in triazine herbicide susceptible biotypes, because of a slight change in a chloroplast protein, triazine resistant biotypes are able to continue normal photosynthesis upon exposure to triazine herbicides (see **Figure 2**). The potential for developing herbicide resistant biotypes is greatest when an herbicide has a single site of action (**Figures 1** and **2**).

Regardless of the mechanism for resistance, becoming familiar with the herbicide mode of action can help design programs that prevent the introduction and spread of herbicide resistant weeds. Management programs for herbicide resistance should emphasize an integrated approach that stresses prevention. Dependence on a single strategy or herbicide family for managing weeds will surely increase the likelihood of additional herbicide resistance problems in the future. Some guidelines for an integrated approach to managing herbicide resistant weeds are given below.

STRATEGIES FOR PREVENTING OR MANAGING HERBICIDE RESISTANCE

- ✓ Practice crop rotation.
- ✓ Rotate herbicide families and use herbicides with different modes of action.
- ✓ Use herbicide mixtures with different modes of action.
- ✓ Control weedy escapes and practice good sanitation to prevent the spread of resistant weeds.
- ✓ Integrate cultural, mechanical, and chemical weed control methods.

Herbicide Families

An understanding of how herbicides kill weeds (i.e., herbicide mode of action) may be useful in selecting and applying the proper herbicide for a given weed control problem and for preventing herbicide resistance problems. Understanding herbicide mode of action is also very useful in diagnosing herbicide injury complaints. Although a large number of herbicides are available in the marketplace, several have similar chemical properties and herbicidal activity. Herbicides with a common chemistry are grouped into “families.” Herbicide families are a convenient way of organizing information about herbicides. In addition, two or more herbicide families may have the same mode of action within the plant and thus express the same herbicide activity and injury symptoms. The following paragraphs describe the characteristics of widely used herbicide families grouped by their mode of action. These seven major modes of action are as follows: growth regulation, amino acid synthesis inhibition, lipid synthesis inhibition, seedling growth inhibition, photosynthesis inhibition, cell membrane disruption, and pigment inhibition.

I. Growth Regulators

1. *Distorted soybean growth (epinasty) due to volatilization, drift, or misapplication of a growth regulator herbicide.*

2. *Soybean callus tissue formation due to early season misapplication of a growth regulator herbicide.*

3. *Parallel veination and strapping of soybean leaves due to 2,4-D drift.*

4. *Cupping of soybean leaves due to dicamba drift.*

I. Growth Regulators

The growth regulators include the following herbicide families: phenoxy acetic acids, benzoic acids, and the pyridines. Growth regulator herbicides can act at multiple sites in a plant to disrupt hormone balance and protein synthesis and thereby cause a variety of plant growth abnormalities. Growth regulator herbicides selectively kill broadleaf weeds; however, they are capable of injuring grass crops. Herbicides in this group can move in both the xylem and the phloem to areas of new plant growth. As a result, many herbicides in this group are effective on perennial and annual leaf weeds. Herbicide uptake is primarily through the foliage but root uptake is possible. Injury symptoms are most obvious on newly developing leaves.

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1. Phenoxy Acetic Acids

a. **Use:** 2,4-D for small grains, corn, grass pastures, and non-cropland

MCPA for small grains

2,4-DB for alfalfa and soybeans

b. **Injury Symptoms:** Broadleaf plants exhibit stem twisting (epinasty), callus tissue formation, and leaf malformations (cupping, crinkling, parallel veins, leaf strapping). Corn plants exhibit rolled leaves (onion-leafing), fused brace roots, stalk bending and brittleness, and missing kernels. Small grains exhibit twisted flag leaves, sterile florets or multiple florets. See **Photos 1** to **10**.

c. **Site of Action:** Specific site(s) unknown, believed to have multiple sites of action.

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***I. Growth Regulators** (Continued)*

5. Fused brace roots may occur if 2,4-D or dicamba are applied at excessive rates or applied beyond the seedling growth stage of corn.

6. Corn onion-leafing due to a late application of 2,4-D or dicamba. Certain hybrids are more susceptible to injury than others.

7. Bent or "elbowed" corn stalks may occur when 2,4-D or dicamba are applied to rapidly growing corn. Injury potential is generally greater with 2,4-D than dicamba.

8. Application of a growth regulator herbicide before the tillering stage of small grain development may result in an elongated rachis, twisted awns, or missing spikelets.

9. Application of 2,4-D or dicamba during the "tassel" to "dough" corn growth stages may result in lack of kernel set or development.

10. Application of a growth regulator herbicide after the jointing stage of small grain development may result in a twisted flag leaf, improper head emergence, and sterile seed heads.

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II. Amino Acid Synthesis Inhibitors

11. Corn stunting with interveinal chlorosis or purpling may result through misapplication or carryover of imidazolinone or sulfonylurea herbicide residues.

12. Carryover of imidazolinone or sulfonylurea residues to corn may inhibit root development by pruning lateral roots.

13. Imidazolinone or sulfonylurea herbicides can stunt soybeans and cause the leaves to appear yellow or golden (chlorotic) in color.

2. Benzoic Acids

a. **Use:** Dicamba (Banvel, Clarity) for corn, wheat, oats, sorghum, pastures, and noncropland

b. **Injury Symptoms:** Banvel injury is similar to that caused by the phenoxy acetic acid herbicides; however, broadleaf plants may exhibit more cupping than strapping of leaf tissue. See **Photo 4.**

c. **Site of Action:** Specific site(s) unknown, believed to have multiple sites of action.

3. Pyridines

a. **Use:** Clopyralid (Stinger) for small grains, sugarbeets, corn, and grass pastures

Picloram (Tordon) for noncropland, small grains, and grass pastures

Triclopyr (Crossbow, see package mixtures, **Table 11**) for noncropland and grass pasture

b. **Injury Symptoms:** Similar to the phenoxy acetic acids. See **Photos 1** to **10**.

c. **Site of Action:** Specific site(s) unknown, believed to have multiple sites of action.

II. Amino Acid Synthesis Inhibitors

The amino acid synthesis inhibitors include the following herbicide families: sulfonylureas, imidazolinones, sulfonamide, and amino acid derivatives. Amino acid synthesis inhibitors act on a specific enzyme to prevent the production of specific amino acids, key building blocks for normal plant growth and development (see **Figure 1**). Sulfonylurea, imidazolinone, and sulfonamide herbicides prevent the production of three essential branch-chain amino acids by inhibiting one key plant enzyme. The amino acid derivative herbicides inhibit the production of three essential aromatic amino acids by inhibiting another key plant enzyme. In general, injury symptoms are slow to develop (1 to 2 weeks) and include stunting or slowing of plant growth and a slow plant death. Herbicides in the sulfonylurea, imidazolinone, and sulfonamide families can move in both the xylem and phloem to areas of new growth and can be taken up through plant foliage and roots. Herbicides in these three families may have activity on annual and perennial broadleaf or grass weeds and may be soil- or foliar-applied. The amino acid derivative herbicides are nonselective and the site of uptake is the plant foliage. Herbicides in this family move via the phloem to all parts of the plant; these are excellent perennial weed control herbicides and are active on annual weeds as well.

1. Imidazolinones

a. **Use:** Imazamethabenz (Assert) for wheat, barley, and sunflowers

Imazaquin (Scepter) for soybeans

Imazethapyr (Pursuit) for soybeans, dry beans, and peas

b. **Injury Symptoms:** Grass plants may be stunted, with interveinal yellowing (chlorosis) or purpling. Corn plants may be stunted and show symptoms of root inhibition such as pruning of lateral roots. Leaves emerging from the corn whorl may not unfurl properly and may be yellow to translucent in appearance. Broadleaf plants may be stunted and chlorotic or purple. Soybean injury can range from stunting to death of the terminal growing point. Soybean leaves may be yellow in appearance and leaf veination may appear red or purple in color. See **Photos 11** to **18**.

1. Imidazolinones (Continued)

c. **Site of Action:** Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

2. Sulfonylureas

a. **Use:** Chlorimuron (Classic) for soybeans

Chlorsulfuron (Glean) for small grains and the Conservation Reserve Program (CRP)

Primisulfuron (Beacon) for corn

Thifensulfuron (Harmony) for small grains
(Pinnacle) for soybeans

Triasulfuron (Amber) for small grains

Nicosulfuron (Accent) for corn

Metsulfuron (Ally) for small grains, grass pastures, and CRP

Tribenuron (Express) for small grains

b. **Injury Symptoms:** Same as the imidazolinone herbicides. See **Photos 11** to **18**.

c. **Site of Action:** Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

3. Sulfonamides

a. **Use:** Flumetsulam (Broadstrike) experimental for corn and soybeans.

b. **Injury Symptoms:** Same as the imidazolinone herbicides. See **Photos 11** to **18**.

c. **Site of Action:** Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

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II. Amino Acid Synthesis Inhibitors *(Continued)*

14. Imidazolinone or sulfonylurea herbicides occasionally can cause soybean leaf veins to appear red or purple. This symptom appears to be unique to this herbicide chemistry.

15. Severe imidazolinone or sulfonylurea injury on soybeans may result in the death of the terminal growing point. Note also the "characteristic" red leaf veination.

16. Severe imidazolinone or sulfonylurea injury on soybeans may result in the death of stem pith tissue. Note also the "characteristic" red leaf veination.

17. Misapplication of imidazolinone or sulfonylurea herbicides onto corn may result in stunted yellow corn, improper leaf unfurling, and translucent leaf tissue.

18. Imidazolinone and sulfonylurea herbicides kill weeds slowly. Injury symptoms are slow to develop (1 to 2 weeks); however, plant growth may slow or stop shortly after herbicide application.

19. Amino acid type herbicides are nonselective. Injury symptoms are slow to appear but occur first on new growth. 19

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4. Amino Acid Derivatives

a. **Use:** Glyphosate (Roundup, Ranger, Rodeo) nonselective weed control for burndown and spot treatments in corn, soybeans, small grains, pasture, and noncropland

b. **Injury Symptoms:** Plant foliage, especially new growth, will first yellow and then turn brown and die within 10 to 14 days after herbicide application. See Photo **19**.

c. **Site of Action:** 5-enolpyruvyl-shikimate-3 phosphate synthase (EPSP synthase) enzyme.

III. Lipid Synthesis Inhibitors

The lipid synthesis inhibitors include the following herbicide families: aryloxyphenoxypropionates and cyclohexanediones.These herbicides prevent the formation of fatty acids, components essential to the production of plant lipids. Lipids are vital to the integrity of cell membranes and to new plant growth. The lipid synthesis inhibitor herbicides inhibit a single key enzyme involved in fatty acid biosynthesis (**Figure 1**). Broadleaf plants are tolerant to these herbicide families, however, almost all perennial and annual grasses are susceptible. Injury symptoms are slow to develop (7 to 14 days) and appear first on new leaves emerging from the whorl of the grass plant. These herbicides are taken up by the foliage and move in the phloem to areas of new growth.

1. Cyclohexanediones

a. **Use:** Sethoxydim (Poast, Poast Plus) for soybeans and alfalfa

Clethodim (Select) for soybeans

b. **Injury Symptoms:** Injury is seen on grass plants only. Newer leaf tissue will be yellow (chlorotic) or brown (necrotic) and the leaves in the leaf whorl can be easily separated from the rest of the plant. See **Photos 20** to **23**.

c. **Site of Action:** Acetyl-CoA carboxylase enzyme.

2. Aryloxyphenoxypropionates

a. **Use:** Diclofop (Hoelon) for small grains

Fluazifop (Fusilade DX, Fusilade 2000) for soybeans

Fenoxaprop (Whip, Option II) for soybeans

Quizalofop (Assure II) for soybeans

b. **Injury Symptoms:** Same as the cyclohexanedione herbicides. See **Photos 20** to **23**.

c. **Site of Action:** Acetyl-CoA carboxylase enzyme.

III. Lipid Synthesis Inhibitors

20. The newly developing leaf tissue slowly turns yellow, then brown, and eventually the plant dies.

21. Leaves in the leaf whorl are easily separated from the plant.

22. Misapplication of sethoxydim onto corn. Note the greatest injury is to the newer leaves.

23. Spray drift of sethoxydim onto corn. Note the yellow to translucent color of the corn leaf.

IV. Seedling Growth Inhibitors

The seedling growth inhibitors include the following herbicide families: dinitroanilines, acetanilides, and thiocarbamates. Seedling growth inhibitors interfere with new plant growth, thereby reducing the ability of seedlings to develop normally in the soil. Herbicides in these families must be soil-applied. Plants can take up these herbicides after germinating until the seedling emerges from the soil. Therefore, these herbicides are only effective on seedling annual or perennial weeds. Plants that have emerged from the soil uninjured are likely to remain unaffected. Seedling growth inhibitors are active at two main sites, the developing shoot and the root. Much more is known about how seedling root inhibiting herbicides work than about how seedling shoot inhibitors work. The root inhibitors stop plant cells from dividing, which inhibits shoot elongation and lateral root formation. Uptake is through developing roots and shoots. Because herbicide movement within the plant is limited, herbicide injury is confined primarily to plant roots and shoots. Shoot inhibiting herbicides are taken up by developing roots and shoots and can move via the xylem to areas of new growth. There is evidence to suggest that these herbicides can affect multiple sites within a plant, primarily interfering with lipid and protein synthesis.

A. Root Inhibitors

1. Dinitroanilines

a. **Use:** Benefin (Balan) for alfalfa

Ethalfluralin (Sonalan) for soybeans

Pendimethalin (Prowl) for corn (preemergence only), soybeans, dry beans, and sunflowers

Trifluralin (Treflan) for soybeans, dry beans, and sunflowers

b. **Injury Symptoms:** General symptoms include stunted plants that do not fully emerge from the soil and short, thick lateral roots. Grass shoots are short and thick and may appear red or purple in color.

Broadleaf plants may have swollen and cracked hypocotyls (the area below the cotyledons). Following preemergence treatments, callus tissue may appear at the base of soybean stems. See **Photos 24** to **27**.

c. **Site of Action:** Tubulin protein involved in cell division.

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IV. Seedling Growth Inhibitors

A. Root Inhibitors

24. Carryover of dinitroaniline herbicide residues or misapplication can cause corn injury. Seedlings may be stunted and roots shortened and thickened.

25. Dinitroaniline induced root inhibition may stunt corn roots, resulting in purple corn. Certain hybrids, compaction, or other stresses that slow plant growth can also result in the purple coloration of corn.

26. Excessive rates of dinitroaniline herbicides may result in seedling soybean injury. Soybean seedlings may have pruned roots and swollen or cracked hypocotyls.

27. Occasionally, a preemergence application or shallow incorporation of dinitroaniline herbicides can cause callus tissue to form on the plant stem, near the soil surface. As a result, the stem is brittle and the plant is very susceptible to lodging.

B. Shoot Inhibitors

1. Acetanilides

a. **Use:** Alachlor (Lasso) for corn, dry beans, sorghum, sunflowers, and soybeans

Acetochlor (Harness Plus, Surpass) for corn

Dimethenamid (Frontier) for corn and soybeans

Metolachlor (Dual) for corn, dry beans, sorghum, and soybeans

Propachlor (Ramrod) for corn, flax, and sorghum

b. **Injury Symptoms:** General symptoms include stunting of shoots that result in abnormal seedlings that do not emerge from the soil. Grasses may leaf-out underground or leaves may not properly unfurl. Broadleaves may have crinkled leaves and/or a shortened mid-vein, which produces a "drawstring" effect. See **Photos 28** and **29**.

c. **Site of Action:** Specific site(s) unknown, believed to have multiple sites of action.

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2. Thiocarbamates

a. **Use:** EPTC (Eptam) for alfalfa, dry beans, flax, sugarbeets, and sunflowers

EPTC plus safener (Eradicane, Eradicane Extra) for corn

Butylate plus safener (Sutan+) for corn

Triallate (Far-Go) for wheat and barley

b. **Injury Symptoms:** General symptoms include stunting of shoots and poor emergence from the soil. Grasses may fail to emerge from the coleoptile or leaf-out underground. Leaf tips may not unfurl from the coleoptile properly, which results in the "buggy whip" effect. Broadleaves may have crinkled or puckered leaves or leaf buds may not open. See **Photos 30** and **31**.

c. **Site of Action:** Specific site(s) unknown, believed to have multiple sites of action.

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B. Shoot Inhibitors

28. Cool wet soils or excessive rates of acetanilide herbicides may cause improper unfurling of corn leaves. Injured plants often outgrow this damage.

29. Acetanilide herbicide injury to soybeans may cause leaf puckering (i.e., the "drawstring effect") where the leaf midrib is shortened.

30. Thiocarbamate herbicides may occasionally cause corn leaves to fail to unfurl properly, resulting in stunted and twisted corn. Certain hybrids, cool wet soils, and excessive herbicide rates can enhance the likelihood of crop injury.

31. Misapplication of a thiocarbamate herbicide onto a soybean field can cause the malformation of leaves and failure of the terminal growing plant to open (i.e., "bud seal").

V. Photosynthesis Inhibitors

The photosynthesis inhibitors include the following herbicide families: triazines, phenylureas, uracils, benzothiadiazoles, and nitriles. Photosynthesis inhibitors shut down the photosynthetic (food producing) process in susceptible plants by binding to specific sites within the plant's chloroplasts (**Figure 2**). Inhibition of photosynthesis could result in a slow starvation of the plant; however, the plant experiences a more rapid death that is believed to be due to the production of secondary toxic substances. Injury symptoms include yellowing (chlorosis) of leaf tissue followed by death (necrosis) of the tissue. Three of the herbicide families (triazines, phenylureas, and uracils) are taken up into the plant via the roots or foliage and move in the xylem to plant leaves. As a result, injury symptoms will first appear on the older leaves, along the leaf margin. After foliar application, triazine, phenylurea, and uracil herbicides are less mobile and do not move out of the leaf tissue. The nitrile and benzothiadiazole herbicide families are not mobile in plants and are classified as postemergence contact herbicides. These herbicides have no soil activity. Contact herbicides must thoroughly cover a susceptible plant's foliage if complete control is to be achieved. Photosynthetic inhibitors may control annual or perennial grass or broadleaf weeds.

A. Mobile Herbicides

1. Triazines

a. **Use:** Ametryn (Evik) for corn

Atrazine for corn and sorghum

Cyanazine (Bladex) for corn

Simazine (Princep) for corn

Metribuzin (Lexone, Sencor) for alfalfa and soybeans

Hexazinone (Velpar) for alfalfa

b. **Injury Symptoms:** Photosynthesis inhibitors do not prevent seedlings from germinating or emerging. Injury symptoms only occur after the cotyledons and first leaves emerge. Initial injury symptoms include yellowing of the leaf margins or tips. In broadleaf plants, yellowing between the leaf veins (interveinal chlorosis) may occur. Older and larger leaves will be affected first because they take up more of the herbicide-water solution and they are the primary photosynthetic tissue of the plant. Injured leaf tissue will eventually turn brown and die. Due to the chemical nature of the herbicide/soil relationship, injury symptoms are likely to increase as the soil pH increases (higher than pH 7.2). See **Photos 32** to **34**.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

32

33

34

V. Photosynthesis Inhibitors

A. Mobile Herbicides

32. Yellowing and browning of corn leaf tissue may result following the application of cyanazine. Injury is most likely following prolonged cool, wet conditions that stress the plant.

33. Interveinal chlorosis (yellowing) and necrosis (browning) of older soybean leaf tissue may result from the use of triazine herbicides such as metribuzin or the carryover of herbicide residues of atrazine or simazine.

34. Wheat is sensitive to atrazine and may be injured from residues that carryover from a previous year. Yellowing and browning of older leaf tissue are the primary injury symptoms.

35

36

B. Nonmobile Herbicides

35. Bentazon leaf burn on soybeans occasionally occurs under stress conditions or very warm temperatures. This injury is cosmetic only, with little risk of yield reduction.

36. Bromoxynil injury on corn demonstrates the contact nature of this herbicide.

2. Phenylureas

a. **Use:** Linuron (Lorox) for soybeans and corn

Tebuthiuron (Spike) for grass pasture and noncropland

b. **Injury Symptoms:** Same as for the triazine herbicides. See **Photos 32** and **33**.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

3. Uracils

a. **Use:** Terbacil (Sinbar) for alfalfa

b. **Injury Symptoms:** Same as for triazine herbicides. See **Photos 32** and **33**.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

B. Nonmobile Herbicides

1. Benzothiadiazoles

a. **Use:** Bentazon (Basagran) for soybeans, corn, dry beans, and grain sorghum

b. **Injury Symptoms:** Plant injury is confined to foliage that has come in contact with the herbicide. Affected leaves will become yellow or bronze in color and eventually turn brown and die. Injury symptoms can look similar to the injury caused by cell membrane disrupters. Crop oil concentrate and other additives may increase weed control and crop injury symptoms. See **Photo 35**.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

2. Nitriles

a. **Use:** Bromoxynil (Buctril) for wheat, barley, oats, rye, flax, corn, and alfalfa

b. **Injury Symptoms:** Plant injury is confined to foliage that has come in contact with the herbicide. Foliage that has been thoroughly covered with the herbicide will turn yellow, then turn brown and die. Contact of a low rate of herbicide with leaves may result in spotting or speckling of the leaf surface. Crop oil concentrates and other additives may intensify injury symptoms. See **Photo 36**.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

3. Pyridazines

a. **Use:** Pyridate (Tough) for corn and peanuts

b. **Injury Symptoms:** Plant injury is confined to foliage that has come in contact with the herbicide. Foliage that has been thoroughly covered with the herbicide will turn yellow, then turn brown and die. Contact of a low rate of herbicide with leaves may result in spotting or speckling of the leaf surface. Crop oil concentrates and other additives may intensify injury symptoms.

c. **Site of Action:** D-1 quinone-binding protein of photosynthetic electron transport.

VI. Cell Membrane Disrupters

The cell membrane disrupters include the diphenylether and bipyridylium herbicide families. These herbicides are postemergence contact herbicides that are activated by exposure to sunlight to form oxygen compounds such as hydrogen peroxide. These oxygen compounds destroy plant tissue by rupturing plant cell membranes. Destruction of cell membranes results in a rapid browning (necrosis) of plant tissue. On a bright and sunny day, herbicide injury symptoms can occur in 1 to 2 hours. Because these are contact herbicides, they are excellent for burndown of existing foliage and postemergence control of annual weeds. Perennial weeds usually regrow because there is no herbicide movement to underground root or shoot systems. These herbicides have little soil activity.

1. Bipyridyliums

a. **Use:** Paraquat (Gramoxone Extra) for nonselective weed control in corn, soybeans, small grains, and dormant alfalfa

Difenzoquat (Avenge) for barley, winter wheat, and some spring and durum wheat varieties

b. **Injury Symptoms:** Plant leaves will have a limp, water-soaked appearance, which is followed by browning (necrosis) of the plant tissue. Drift injury will appear as speckling on leaf tissue. See **Photo 37**.

c. **Site of Action:** Activated by photosystem I (PSI).

2. Diphenylethers

a. **Use:** Acifluorfen (Blazer) for soybeans

Lactofen (Cobra) for soybeans

Fomesafen (Reflex) for soybeans

b. **Injury Symptoms:** Plant leaves will yellow and then turn brown and die. Reddish-colored spotting on the leaf surface may appear shortly after the herbicide is applied. Plants that do not die may be stunted for a week or more. Crop oils and other additives, as well as extremely cool or warm temperatures, may increase plant injury. See **Photo 38**.

c. **Site of Action:** Inhibition of protoporphyrinogen oxidase (Protox).

37

38

VI. Cell Membrane Disrupters

37. Paraquat is a nonselective postemergence herbicide that kills plant tissue quickly. Injury symptoms can be seen within several hours after application, first as water-soaked areas on the leaf that eventually turn brown.

38. Diphenylether herbicides may cause spotting of the leaf tissue that is contacted by the herbicide spray. The likelihood of soybean recovery from this type of injury is relatively good.

39

40

41

VII Pigment Inhibitors

39. Corn injury from clomazone can occur through misapplication, drift, or carryover. Shortly after corn emergence, the corn plant may appear yellow, then bleached white.

40. Small grains such as wheat are sensitive to clomazone residues. Plants appear yellow or white.

41. Preemergence applications of clomazone may move off-target by particle or vapor drift. Sensitive ornamental and commercially grown plants that come in contact with the clomazone drift may appear yellow or white, especially the new vegetative growth. Unless the injury is severe, plants generally recover from the clomazone injury.

VII. Pigment Inhibitors

Pigment inhibitors prevent plants from forming photosynthetic pigments. As a result, the affected plant parts become white to translucent. Clomazone (Command), a soil-applied herbicide, is the only member of the isoxazolidinone family in use at this time. Command is taken up by plant roots and shoots and can move in the xylem to plant leaves. The newly developed foliage of many plant species is so sensitive to Command that very small amounts can whiten new plant growth. Norflurazon (Zorial), a soil-applied herbicide, is the only member of the pyridazinone family in use at this time. Zorial is taken up by plant roots and moves to the growing points of susceptible plants. Susceptible weeds will emerge as white plants before dying.

1. Isoxazolidinones

a. **Use:** Clomazone (Command) for soybeans

b. **Injury Symptoms:** Plants turn white, often becoming translucent at the leaf tips. In corn, if more than 75% of the plant is white it will likely die. See **Photos 39** to **41**.

c. **Site of Action:** Specific site(s) unknown but is different than the pyridazinones.

2. Pyridazinones

a. **Use:** Norflurazon (Zorial) for soybeans and cotton grown in the southern U.S.A. only.

b. **Injury Symptoms:** Plants turn white, often becoming translucent.

c. **Site of Action:** Phytoene and phytofluene desaturase enzymes of the terpenoid pathway.

Table 2. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as growth regulators.*

Site of Action = Inhibition of Auxin Transport

Chemical Family	Common Name	Trade Name	Package Mix
Semicarbazone	diflufenzopyr	Celebrity Plus	Yes
		Distinct	Yes
		Overdrive	Yes

Site of Action = Synthetic Auxins

Chemical Family	Common Name	Trade Name	Package Mix
Benzoic acid	dicamba	Banvel	No
		Brash	Yes
		Celebrity Plus	Yes
		Cimarron Max	Yes
		Clarity	No
		Distinct	Yes
		Marksman	Yes
		Northstar	Yes
		Overdrive	Yes
		Rave	Yes
		Sterling	No
		Sterling Plus	Yes
		Weedmaster	Yes
		Yukon	Yes
Phenoxy carboxylic acid	2,4-D	2,4-D, others	No
		Brash	Yes
		Cimarron Max	Yes
		Crossbow	Yes
		Curtail	Yes
		Grazon P+D	Yes
		Saber	No
		Salvo	No
		Shotgun	Yes
		Starane + Saber	Yes
		Starane + Salvo	Yes
		Weedmaster	Yes
	2,4-DB	Butoxone	No
		Butyrac	No
	MCPA	Bison	Yes
		Bison Advanced	Yes
		Bronate Advanced	Yes
		Curtail M	Yes
		MCPA, others	No
		Starane + Sword	Yes
		Sword	No
Pyridine carboxylic acid	aminopyralid clopyralid	Milestone	No
		Accent Gold WDG	Yes
		Curtail	Yes
		Curtail M	Yes
		Hornet	Yes
		Redeem	Yes
		Stinger	No
		Stinger	No
		WideMatch	Yes
	fluroxypyr	Starane	No
		Starane + Saber	Yes
		Starane + Salvo	Yes
		Starane + Sword	Yes
		WideMatch	Yes
	picloram	Grazon P+D	Yes
		Tordon 22K	No
	triclopyr	Crossbow	Yes
		Garlon	No
		Redeem	Yes
		Remedy	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 3. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as amino acid synthesis (ALS enzyme) inhibitors.*

Site of Action = Inhibition of Acetolactate Synthase ALS

Chemical Family	Common Name	Trade Name	Package Mix
Imidazolinone	imazamethabenz	Assert	No
	imazamox	Beyond	No
		Raptor	No
	imazapic	Plateau	No
	imazapyr	Lightning	Yes
	imazethapyr	Extreme	Yes
		Lightning	Yes
		Pursuit	No
		Pursuit Plus	Yes
Sulfonylaminocarbonyltriazolinone	flucarbazone	Everest	No
Sulfonylurea	chlorimuron	Classic	No
		Synchrony XP	Yes
	foramsulfuron	Equip	Yes
		Option	No
	halosulfuron	Permit	No
		Yukon	Yes
	iodosulfuron	Equip	Yes
	mesosulfuron	Rimfire	Yes
		Silverado	No
	metsulfuron-methyl	Cimarron	No
		Cimarron Max	Yes
	nicosulfuron	Accent	No
		Accent Gold WDG	Yes
		Basis Gold	Yes
		Celebrity Plus	Yes
		Steadfast	Yes
		Steadfast ATZ	Yes
		Stout	Yes
	primisulfuron	Beacon	No
		Northstar	Yes
	rimsulfuron	Accent Gold WDG	Yes
		Basis	Yes
		Basis Gold	Yes
		Matrix	No
		Resolve	No
		Steadfast	Yes
		Steadfast ATZ	Yes
	thifensulfuron	Affinity BroadSpec	Yes
		Affinity TankMix	Yes
		Basis	Yes
		Harmony Extra XP	Yes
		Harmony GT XP	No
		Stout	Yes
		Synchrony XP	Yes
	triasulfuron	Rave	Yes
	tribenuron	Affinity BroadSpec	Yes
		Affinity TankMix	Yes
		Express XP	No
		Harmony Extra XP	Yes
	triflusulfuron	UpBeet	No
Triazolopyrimidine	chloransulam-methyl	FirstRate	No
	flumetsulam	Accent Gold WDG	Yes
		Hornet	Yes
		Python	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 4. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as amino acid synthesis (EPSP enzyme) inhibitors.*

Site of Action = Inhibition of EPSP Synthase

Chemical Family	Common Name	Trade Name	Package Mix
Glycine	glyphosate	Cornerstone	No
		Durango	No
		Extreme	Yes
		Field Master	Yes
		Glyphomax XRT	No
		Roundup	No
		Roundup Original MAX	No
		Roundup UltraMAX II	No
		Roundup WeatherMAX	No
		Touchdown	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 5. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as ammonia assimilation inhibitors.*

Site of Action = Inhibition of Glutamine Synthetase

Chemical Family	Common Name	Trade Name	Package Mix
Phosphinic acid	glufosinate	Liberty	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 6. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as lipid synthesis inhibitors.*

Site of Action = Inhibition of Acetyl CoA Carboxylase (ACCase)

Chemical Family	Common Name	Trade Name	Package Mix
Aryloxyphenoxypropionate	clodinafop-propargyl	Discover	No
		Discover NG	No
	fenoxaprop	Fusion	Yes
		Puma	No
	fluazifop-P	Fusilade DX	No
		Fusion	Yes
	quizalofop	Assure II	No
		Targa	No
Cyclohexanedione	clethodim	Arrow	No
		Prism	No
		Section	No
		Select	No
		Select Max	No
	sethoxydim	Poast	No
		Poast Plus	No
		Rezult	Yes
	tralkoxydim	Achieve	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 7. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as seedling shoot inhibitors.*

Site of Action = Inhibition of Cell Division

Chemical Family	Common Name	Trade Name	Package Mix
Chloroacetamide	acetochlor	Confidence	No
		Confidence Xtra	Yes
		Confidence Xtra 5.6L	Yes
		Degree	No
		Degree Xtra	Yes
		Double Team	Yes
		Field Master	Yes
		First Act	No
		Fultime	Yes
		Harness	No
		Harness Xtra	Yes
		Harness Xtra 5.6L	Yes
		Keystone	Yes
		Keystone LA	Yes
		Surpass	No
		Topnotch	No
	alachlor	Bullet	Yes
		Intrro	No
		Lariat	Yes
		Lasso	No
		Micro Tech	No
	dimethenamid-P	G-Max Lite	Yes
		Outlook	No
		Propel	No
	metolachlor	Parallel PCS	No
		Parallel Plus	Yes
	metolachlor & benoxacor	Parallel	No
	pronamide	Kerb	No
	s-metolachlor	Bicep Lite II Magnum	Yes
		Boundary 6.5	Yes
		Camix	Yes
		Charger MAX	No
		Charger MAX ATZ	Yes
		Charger MAX ATZ Lite	Yes
		Cinch	No
		Cinch ATZ	Yes
		Cinch ATZ Lite	Yes
		Dual II Magnum	No
		Dual Magnum	No
		Lumax	Yes
Oxyacetamide	flufenacet	Define	No

Site of Action = Inhibition of Lipid Synthesis (not ACCase inhibition)

Chemical Family	Common Name	Trade Name	Package Mix
Benzofuran	ethofumesate	BnB Plus	Yes
		Des-Phen-Etho	Yes
		Etho SC	No
		Ethotron SC	No
		Nortron SC	No
		Progress	Yes
Thiocarbamate	cycloate	Ro-Neet	No
	EPTC	Eptam	No
	triallate	Fargo	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 8. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as seedling root inhibitors.*

Site of Action = Microtubule Assembly Inhibition

Chemical Family	Common Name	Trade Name	Package Mix
Dinitroanilines	benefin	Balan	No
	ethalfluralin	Sonalan	No
	pendimethalin	Pendant	No
		Pendimax	No
		Prowl	No
		Prowl H20	No
		Pursuit Plus	Yes
	trifluralin	Treflan	No
		Trust	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 9. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as cell membrane disrupters.*

Site of Action = Inhibition of Photosystem I - Electron Diversion

Chemical Family	Common Name	Trade Name	Package Mix
Bipyridylium	paraquat	Gramoxone	No

Site of Action = Inhibition of Protoporphyrinogen Oxidase (PPO)

Chemical Family	Common Name	Trade Name	Package Mix
Diphenylether	acifluorfen	Storm	Yes
		Ultra Blazer	No
	fomesafen	Flexstar	No
		Reflex	No
	lactofen	Cobra	No
		Phoenix	No
N-phenylphtalimide	flumiclorac	Resource	No
	flumioxazin	Valor	No
Triazolinone	carfentrazone-ethyl	Aim	No
		Avalanche	No
	propoxycarbazone	Rimfire	Yes
	sulfentrazone	Spartan	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 10. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as pigment inhibitors.*

Site of Action = Bleaching: Inhibition of 4-Hyroxyphenl-Pyruvate-Dioxygenase (4-HPPD)

Chemical Family	Common Name	Trade Name	Package Mix
Callistemone	mesotrione	Callisto	No
		Camix	Yes
		Lumax	Yes

Site of Action = Bleaching: Inhibition of Diterpenes

Chemical Family	Common Name	Trade Name	Package Mix
Isoxazolidinone	clomazone	Command	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 11. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as Photosynthesis (mobile) inhibitors.*

Site of Action = Inhibition of Photosynthesis at Photosystem II (Binding Behavior A)

Chemical Family	Common Name	Trade Name	Package Mix
Phenyl-carbamate	desmedipham	Alphanex	No
		Betamix	Yes
		Betanex	No
		BnB Plus	Yes
		Des	No
		Des-Phen-Etho	Yes
		DP-Mix	Yes
		Phen-Des 8+8	Yes
		Progress	Yes
		Progress	Yes
	phenmedipham	Betamix	Yes
		BnB Plus	Yes
		Des-Phen-Etho	Yes
		DP-Mix	Yes
		Phen-Des 8+8	Yes
		Progress	Yes
Triazine	atrazine	atrazine, others	No
		Basis Gold	Yes
		Bicep Lite II Magnum	Yes
		Buctril + atrazine	Yes
		Bullet	Yes
		Charger MAX ATZ	Yes
		Charger MAX ATZ Lite	Yes
		Cinch ATZ	Yes
		Cinch ATZ Lite	Yes
		Confidence Xtra	Yes
		Confidence Xtra 5.6L	Yes
		Degree Xtra	Yes
		Double Team	Yes
		Field Master	Yes
		Fultime	Yes
		G-Max Lite	Yes
		Harness Xtra	Yes
		Harness Xtra 5.6L	Yes
		Keystone	Yes
		Keystone LA	Yes
		Laddock S-12	Yes
		Lariat	Yes
		Lumax	Yes
		Marksman	Yes
		Parallel Plus	Yes
		Shotgun	Yes
		Steadfast ATZ	Yes
		Sterling Plus	Yes
	simazine	Princep	No
Triazinone	hexazinone	Velpar	No
		Velpar AlfaMax MP	Yes
	metribuzin	Boundary 6.5	Yes
		Sencor	No
Uracil	terbacil	Sinbar	No

Site of Action = Inhibition of Photosynthesis at Photosystem II (Binding Behavior C)

Chemical Family	Common Name	Trade Name	Package Mix
Amide	propanil	Stampede	No
Urea	diuron	Velpar AlfaMax MP	Yes
	linuron	Lorox	No
	tebuthiuron	Spike	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Table 12. *Cross reference list of chemical family, herbicide common names, and trade[1] names classified as Photosynthesis (non-mobile) inhibitors.*

Site of Action = Inhibition of Photosynthesis at Photosystem II (Binding Behavior B)

Chemical Family	Common Name	Trade Name	Package Mix
Benzothiadiazole	bentazon	Basagran	No
		Laddock S-12	Yes
		Rezult	Yes
		Storm	Yes
Nitrile	bromoxynil	Bison	Yes
		Bison Advanced	Yes
		Bronate Advanced	Yes
		Buctril	No
		Buctril + atrazine	Yes
		Moxy	No

[1] Reference to commercial products or trade names is made with the understanding that no discrimination is intended and no endorsement by the Minnesota Extension Service is implied.

Glossary

Callus tissue — A mass of plant cells that form at a wounded surface.

Chloroplast — A membrane-enclosed structure that contains the green pigment molecules (chlorophyll) essential for photosynthesis (i.e., food production).

Chlorosis — A yellowing in plant color due to a decline in chlorophyll levels.

Contact herbicide — A general classification for herbicides that are unable to move within a plant. A contact herbicide's effectiveness is highly dependent upon uniform coverage of treated soil or plant tissue.

Epinasty — A bending of plant parts (e.g., stems or leaf petioles) downwards due to increased growth on the upper side of an affected plant part. Often associated with the plant growth regulator herbicides.

Herbicide mode of action — The sequence of events from absorption of the herbicide into the plant through plant death. Refers to all plant-herbicide interactions.

Herbicide site of action — The primary biochemical site that is affected by the herbicide, ultimately resulting in the death of the plant. Also referred to as herbicide mechanism of action.

Necrosis — The death of specific plant tissue while the rest of the plant is still alive. Necrotic areas are generally dark brown in color.

Phloem — Plant tissue that functions as a conduit for the movement (translocation) of sugars and other plant nutrients.

Postemergence application — A time of herbicide application occurring after the crop and weeds emerge from the soil. Also referred to as a foliar application.

Preemergence application — A time of herbicide application occurring after a crop is planted but before the crop or weeds emerge from the soil.

Preplanting application — A time of herbicide application occurring before the crop is planted. Often followed by an incorporation (mechanical mixing) into the top 1 to 2 inches of soil. Often referred to as a preplant incorporation treatment.

Systemic herbicide — A general classification for herbicides that are able to move away from the site of absorption to other parts of the plant.

Translocation — The movement of water, plant sugars, and nutrients, herbicides, and other soluble materials from one plant part to another.

Translucent — An absence of leaf tissue pigments that results in the diffusion of light, giving the plant an off-white color.

Xylem — Plant tissue that functions as a conduit for the upward movement (translocation) of water from the roots to above-ground plant parts.

BU-3832-S
Reviewed 1999

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Site of Action References

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Produced by Communication and Educational Technology Services, University of Minnesota Extension Service.