3832 (Rev. 1991)

North Central Regional
Extension Publication 377

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 thirteen 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.

University of Illinois
69 Mumford Hall
1301 W. Gregory Drive
Urbana, IL 61801
217-333-2007

Iowa State University
112 Printing & Publ. Bldg.
Ames, IA 50011-1050
515-294-5247

Michigan State University
10B Ag Hall
East Lansing, MI 48824-1039
517-335-0240

*University of Minnesota
3 Coffey Hall
St. Paul, MN 55108
612-625-8173

*Publishing state

University of Nebraska
Dept. of Ag Communications
Lincoln, NE 68583-0918
402-472-3023

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July, 1991

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 in order 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 plant shoots 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 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, 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.

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

Herbicide Family	Herbicides	Weeds	Country
Dinitroaniline	Trifluralin	Goosegrass	USA
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

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 (acetolactate 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.

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 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 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 destruction, and pigment inhibition.

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 broadleaf weeds. Herbicide uptake is primarily through the foliage but root uptake is possible. Injury symptoms are most obvious on newly developing leaves.

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.

2. Benzoic Acids

a. Use: Dicamba (Banvel) 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.

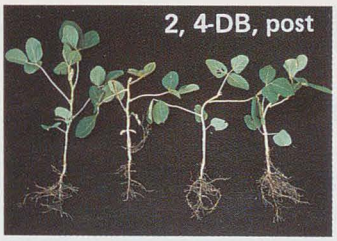
c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

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 2) 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.



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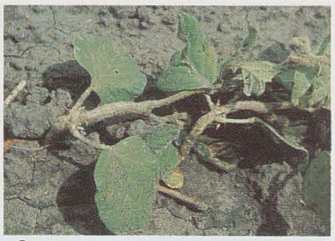
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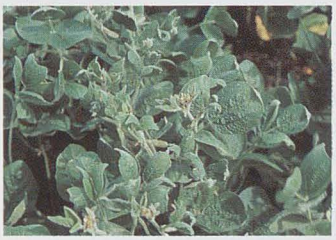
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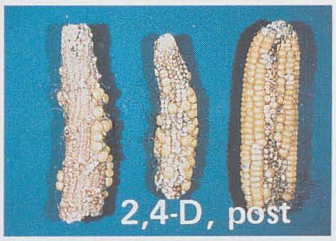

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1. Distorted soybean growth (epinasty) due to volatilization, drift, or misapplication of a growth regulator herbicide.
2. Soybean callus tissue formation due to early season misapplication of a growth regulator herbicide.
3. Parallel veination and strapping of soybean leaves due to 2,4-D drift.
4. Cupping of soybean leaves due to dicamba drift.
5. Corn onion leafing due to a late application of 2,4-D or dicamba. Certain hybrids are more susceptible to injury than others.
6. 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.
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 2,4-D or dicamba during the "tassel" to "dough" corn growth stages may result in lack of kernel set or development.
9. 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.
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.

II. Amino Acid Synthesis Inhibitors

The amino acid synthesis inhibitors include the following herbicide families: sulfonylureas, imidazolinones, 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 and imidazolinone 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 and imidazolinone 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 two 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.

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


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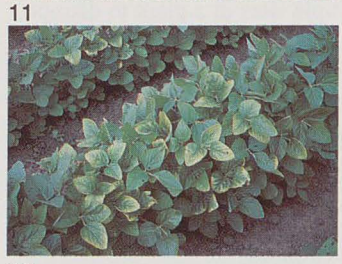
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11. Corn stunting with interveinal chlorosis or purpling may result through misapplication or carryover of imidazolinone or sulfonylurea herbicide residues.
12. Carryover of imidazolinone or sulfonylurea herbicide residues to corn may inhibit root 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.
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 applicaiton.
19. Amino acid type herbicides are nonselective. Injury symptoms are slow to appear but occur first on new growth.

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. 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 Inhibitors

The lipid inhibitors include the following herbicide families: aryloxyphenoxypropionates and cyclohexanediones. These herbicides prevent the formation of fatty acids, components essential for the production of plant lipids. Lipids are vital to the integrity of cell membranes and to new plant growth. The lipid 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) experimental 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.

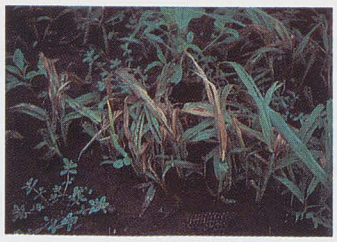
c.Site of Action: Acetyl-CoA carboxylase enzyme.

2. Aryloxyphenoxypropionates

a. Use: Diclofop (Hoelon) for small grains
Fluazifop (Fusilade) for soybeans
Fenoxaprop (Whip, Option) 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.



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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.

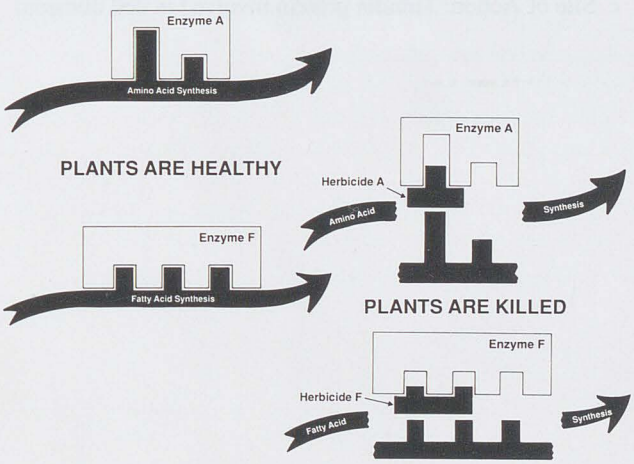

Figure 1. Enzymes - Enzymes function as steps in biological processes. Enzymes are also extremely specialized in their function. As a result, many different enzymes are involved with the many different biological processes that occur within a plant. Some herbicides can stop specific enzymes from functioning, 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.

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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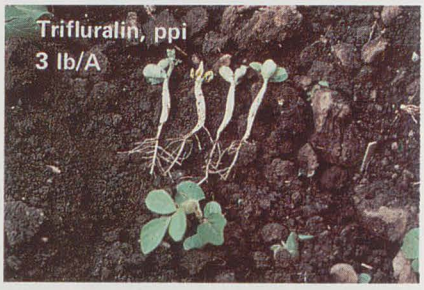

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24. Carryover of dinitroaniline herbicide residues or misapplication can cause corn injury. Seedlings may be stunted and roots shortened and thickened.

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.

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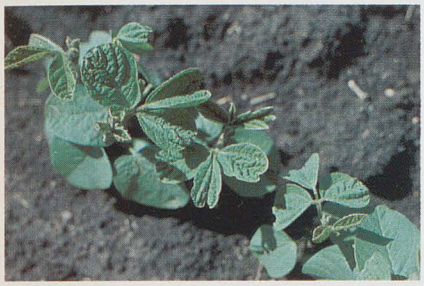

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28. Cool wet soils or excessive rates of acetanilide herbicides may cause improper unfurling of corn leaves. Injured plants often out-grow this damage.

29. Acetanilide herbicide injury to soybeans may cause leaf puckering (ie. 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 (ie. "bud seal").

B. Shoot Inhibitors

1. Acetanilides

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

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.

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 plants 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.

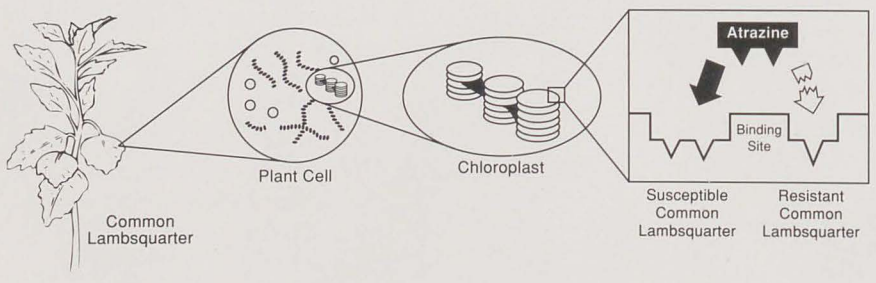

Figure 2. Photosynthetic Inhibitors - The photosynthetic process occurs within a plant cells chloroplasts. Certain herbicides can inhibit photosynthesis by binding to specific sites within the chloroplast. The relationship of a herbicide to the chloroplst binding site is very specific and any modification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

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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 trazine 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.

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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.

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. (See accompanying text on page 12.)

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. Effected 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.

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 on page 11.

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).

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.

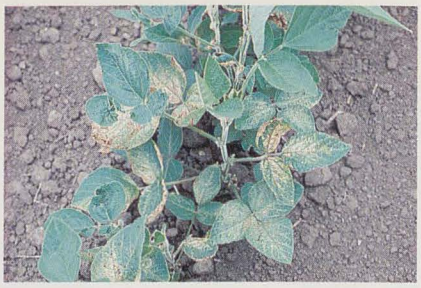
c. Site of Action: Specific site(s) unknown but is different than the pyridazinones.

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



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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. 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.

Table 2. Cross reference list of herbicide trade names and common names, including package mixtures.

Trade[1] Name	Common Name
Accent	Nicosulfuron
Ally	Metsulfuron
Amber	Triasulfuron
Arena[2]	Alachlor
Assert	Imazamethabenz
Assure II	Quizalofop
Atrazine[2]	Atrazine
Avenge	Difenzoquat
Balan	Benefin
Banvel	Dicamba
Basagran	Bentazon
Basalin	Fluchloralin
Beacon	Primisulfuron
Bicep	Atrazine + Metolachlor
Bladex[2]	Cyanazine
Blazer	Acifluorfen
Bronate[2]	Bromoxynil + MCPA
Bronco[2]	Alachlor + Glyphosate
Buckle	Triallate + Trifluralin
Buctril[2]	Bromoxynil
Buctril-Atrazine[2]	Bromoxynil + Atrazine
Bullet[2]	Alachlor + Atrazine
Atrazine	Atrazine
Butyrac	2,4-DB
Cannon[2]	Alachlor + Trifluralin
Canopy	Chlorimuron + Metribuzin
Carbyne	Barban
Classic	Chlorimuron
Cobra	Lactofen
Command	Clomazone
Commence	Clomazone + Trifluralin
Confidence[2]	Alachlor
Cropstar[2]	Alachlor
Crossbow	Triclopyr + 2,4-D ester
Curtail	Clopyralid + 2,4-D amine
Curtail M	Clopyralid + MCPA ester
Cycle[2]	Metolachlor + Cyanazine
Cyclone[2]	Paraquat
Dual	Metolachlor
Eptam	EPTC
Eradicane	EPTC + Dichlormid
Eradicane Extra	EPTC + Dichlormid + Dietholate
Evik	Ametryn
Express	Tribenuron
Extrazine II[2]	Cyanazine + Atrazine
Fallow Master	Glyphosate + Dicamba
Far-Go	Triallate
Freedom[2]	Alachlor + Trifluralin
Fusilade 2000	Fluazifop
Galaxy	Bentazon + Acifluorfen
Gemini	Linuron + Chlorimuron
Glean	Chlorsulfuron
Gramoxone Extra[2]	Paraquat
Harmony	Thifensulfuron
Harmony Extra	Tribenuron + Thifensulfuron
Hoelon[2]	Diclofop
Honcho	Glyphosate
Hyvar XL	Bromacil
Judge[2]	Alachlor
Jury	Glyphosate
Laddock	Bentazon + Atrazine
Landmaster	Glyphosate + 2,4-D
Lariat[2]	Alachlor + Atrazine
Lasso[2]	Alachlor
Lasso + Atrazine[2]	Alachlor + Atrazine
Lexone	Metribuzin
Linex	Linuron
Lorox	Linuron
Lorox Plus	Linuron + Chlorimuron
Marksman[2]	Dicamba + Atrazine
Matrix	Tribenuron + Thifensulfuron
Mirage	Glyphosate
MCPA Amine, others	MCPA Amine
MCPA Ester,others	MCPA Ester
Option	Fenoxaprop
Passport	Trifluralin + Imazethapyr
Pinnacle	Thifensulfuron
Poast	Sethoxydim
Poast Plus	Sethoxydim
Preview	Metribuzin + Chlorimuron
Princep	Simazine
Prowl	Pendimethalin
Prozine[2]	Pendimethalin + Atrazine
Pursuit	Imazethapyr
Pursuit Plus	Imazethapyr + Pendimethalin
Ramrod	Propachlor
Ramrod + Atrazine[2]	Propachlor + Atrazine
Ranger	Glyphosate
Rattler	Glyphosate
Reflex	Fomesafen
Rescue	Naptalam + 2,4-DB
Rodeo	Glyphosate
Roundup	Glyphosate
Ruler	Glyphosate
Salute	Trifluralin + Metribuzin
Scepter	Imazaquin
Select	Clethodim
Sencor	Metribuzin
Show-Off	Glyphosate
Silhouette	Glyphosate
Sinbar	Terbacil
Sonalan	Ethalfluralin
Spike	Tebuthiuron
Squadron	Imazaquin + Pendimethalin
Stall[2]	Alachlor
Stampede CM	Propanil + MCPA
Stinger	Clopyralid
Storm	Acifluorfen + Bentazon
Sutan +	Butylate + Dichlormid
Sutazine[2]	Butylate + Dichlormid + Atrazine
Tiller	Fenoxaprop + 2,4-D + MCPA
Tordon 22K[2]	Picloram
Tornado	Fluazifop + Fomesafen
Treflan	Trifluralin
Trific	Trifluralin
Trillin	Trifluralin
Tri-Scept	Imazaquin + Trifluralin
Turbo	Metolachlor + Metribuzin
2,4-D Amine,others	2,4-D Amine
2,4-D Ester,others	2,4-D Ester
2,4-DB	2,4-DB
Velpar	Hexazinone
Whip	Fenoxaprop
Zorial	Norflurazon

[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.

[2] Restricted Use Herbicide

Herbicide Mode of Action and Injury Symptoms

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Site of Action References

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Further Information

Color photographs available from North Central Regional Publications:
Breneman, D. and R. Behrens. *Herbicide Symptoms in Corn.* 1986 (revised).
North Central Regional Extension Publication 94

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).

Chlorsis - 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.

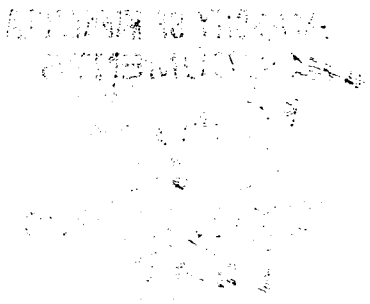
Systemic herbicide - A general classification for herbicides that are able to move away from the site of absorption to other parts of the plant.

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 to serve as a conduit for the upward movemet (translocation) of water from the roots to above-ground plant parts.

AG-FO-3832-E
Revised 1991



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