Herbicide Mode of Action and Sugarbeet Injury Symptoms

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Alan G. Dexter

Extension Sugarbeet Specialist, Department of Crop and Weed Sciences North Dakota State University and the University of Minnesota

Jeffrey L. Gunsolus

Extension Agronomist-Weed Control, Department of Agronomy and Plant Genetics University of Minnesota

William S. Curran Extension Agronomist-Weed Control, Department of Agronomy Pennsylvania State University

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Herbicide Mode of Action

Herbicide effectiveness depends on 1) adequate plant contact; 2) absorption by plants; 3) translocation (i.e. movement) within the plants to the site of action, without deactivation; and 4) toxic levels at the site of action. The application method, whether preplant incorporated, preemergence, or postemergence, largely determines when the herbicide will contact plants and the portion of the plant contacted.

Mode of action refers to the sequence of events from absorption into plants to plant death. The mode of herbicide action may determine the application method needed for best results. For example, herbicides which affect photosynthesis but have little soil residual, such as desmedipham (Betanex) or paraquat (Gramoxone Extra), need to be applied postemergence to leaf tissue. Seedling growth inhibitors, such as trifluralin (Treflan) and EPTC (Eptam), need to be applied to the soil to effectively control newly germinating seedlings.

Soil-Applied Herbicide Phytotoxicity in Plants

Seeds of many weed species are quite small and germinate only 0.5 to 1.0 inch below the soil surface, so soil-applied herbicides should be concentrated in the top 1 to 2 inches of soil for best weed control. Herbicide positioning can be

accomplished by mechanical incorporation or rainfall. Close contact between herbicide and plant is needed for absorption through the roots or shoots and effective weed control. Herbicide absorption through roots will continue as long as the absorbing region near the root tips remains in contact with the herbicide-treated soil. As the roots grow deeper, herbicide uptake declines. Therefore, plants may survive if the root tips grow out of the herbicide-treated soil before herbicide absorption is sufficient to kill the plants.

Many soil-applied herbicides are absorbed through unemerged plant shoots, and plants may be killed or injured before emergence. Volatile herbicides such as the thiocarbamates (EPTC [Eptam] for example) the dinitroanilines (such as trifluralin [Treflan]) can move in the soil and penetrate plant shoots as gasses or liquids. Less volatile herbicides such as the acetanilides (metolachlor [Dual]) probably are absorbed into the shoot only 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 translocation within a plant. The soil-applied dinitroaniline herbicides (such as trifluralin [Treflan]) are not mobile within the plant. Therefore, their primary injury symptoms are mostly confined to the site of uptake. Other herbicides are mobile within the plant, and injury symptoms will generally be most prominent at the site where mobile herbicides concentrate. For example, soil-applied atrazine is absorbed by plant roots and moves upward within the water transport system of the plant to the leaves, where symptoms occur.

Postemergence Herbicide Phytotoxicity in Plants

Effective weed control from postemergence herbicides depends on adequate contact with above-ground plant shoots and leaves. Therefore, the combination of spray nozzle pressure and volume should be selected to obtain necessary plant coverage and drift control.

Weed control from readily translocated herbicides is affected little by changes in droplet size within a normal range. Relatively large spray droplets may result in less weed control than small spray droplets because of poorly translocated herbicides. Small spray droplets give more complete leaf coverage and are retained more than large droplets on hard-to-wet leaves such as vertical, waxy or small leaves. Large spray droplets will better penetrate a spray canopy and drift less than small droplets. Droplet size will be increased by reducing spray pressure, increasing nozzle orifice size, special drift reduction nozzles, additives that increase spray viscosity, and rearward nozzle orientation on aircraft.

Postemergence herbicide rate of uptake and amount absorbed often is determined by the chemical and physical relationships between the leaf surface and the herbicide. Factors such as plant size and age, water stress, air temperature, relative humidity, and adjuvants can influence the rate and amount of herbicide uptake. Adjuvants such as oil concentrates, methylated seed oils, surfactants, or liquid fertilizer solutions can increase herbicide uptake by a plant. Hot and dry conditions, old weeds and weeds under drought stress all can reduce herbicide uptake. The amount and rate of herbicide uptake influence the potential for crop injury and weed control and often explain the year-to-year variation in the effectiveness of herbicides. Also, rapid herbicide absorption by plants will reduce the time for possible removal by rain or degradation by sunlight.

Postemergence herbicides, like soil-applied herbicides, differ in movement 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) generally move both upward and downward within the food transport system 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 may rapidly degrade or deactivate a herbicide to escape that herbicide's toxic effects. For example, corn quickly deactivates atrazine by binding to naturally occurring plant chemicals. Soybean tolerance to metribuzin (Sencor, Lexone) is at least partially due to the deactivation of the herbicide by conjugating (binding) to plant sugar molecules. Sugarbeet

avoids injury from desmedipham (Betanex) partially through rapid metabolism.

Situations may occur in which a crop is injured by a herbicide that is normally not toxic to the crop. 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 desmedipham (Betanex) injury to sugarbeet under hot and wet weather conditions is a good example of environmentally induced herbicide injury. An excessive amount of herbicide due to misapplication also can injure a tolerant crop by overwhelming the crop's herbicide degradation and deactivation systems.

Herbicide Families

An understanding of how herbicides kill weeds (herbicide mode of action) is useful in selecting and applying the proper herbicide for a given weed control problem and for management of weed resistance to herbicides. Herbicide mode of action information also is useful in diagnoses of injury from herbicides.

Although a large number of herbicides are available in the marketplace, they can be divided into groups with similar chemical and phytotoxic properties. Herbicides with a common chemistry have been conveniently organized into "families." In addition, two or more herbicide families may have the same mode of phytotoxic action and thus express the same injury symptoms.

The following sections describe the characteristics of widely used herbicide families grouped by mode of action. These seven major modes of action are: growth regulation, amino acid synthesis inhibition, lipid synthesis inhibition, seedling growth inhibition, photosynthesis inhibition, cell membrane disruption, and pigment inhibition.

I. Growth Regulators

The growth regulators include 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 control broadleaf weeds and can injure sugarbeet. 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 may also occur.

1. Phenoxy Acetic Acids

- a. Use: **2,4-D** for small grains, corn, grass pastures, and noncropland **MCPA** for small grains and grass establishment. **2,4-DB** for alfalfa and soybean
- b. Injury Symptoms: All phenoxy acetic acids produce identical symptoms in sugarbeet. Sugarbeet leaves will be flat on the ground within a few hours after exposure and leaves may remain more prostrate than normal for the rest of the growing season if injury is severe (Photo 1). Leaf petioles exhibit twisting, also called epinasty (Photo 2). Sugarbeet exposed to phenoxy acetic acids in the cotyledon to early four-leaf stage may develop fused petioles and a symptom called "celery stalking" or "trumpeting" (Photo 3). Phenoxy acetic acid herbicides on larger sugarbeet will not produce "celery stalking." New leaf growth generally will resume at about six to 10 days after exposure to phenoxy acetic acids. New leaves often will be malformed with crinkled leaf margins, parallel veins, or leaf strapping. Rapid initiation of new leaf growth suggests less sugarbeet injury and less sugarbeet yield loss than a delayed initiation of new leaf growth. Sugarbeet injured with phenoxy acetic acids should be processed immediately after harvest because injured sugarbeet roots do not store as well as non-injured roots.
- c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

<u>Photo 1</u>. Sugarbeet plants on the right were accidentally treated with 2,4-D. Treated plants are lying more prostrate than normal. (31KB color photo)

Photo 2. Sugarbeet plants were treated with 2,4-D at 0.06 lb/A six days before the photo. Petiole twisting (epinasty) is evident. (15KB color photo)

Photo 3. Sugarbeet was treated in the two-leaf stage with 2,4-D at 0.06 lb/A. Sugarbeet plant has the "celery stalking" or "trumpeting" symptom. (13KB color photo)

2. Benzoic Acids

- a. Use: **Dicamba (Banvel)** for corn, wheat, oats, sorghum, pastures, and noncropland.
- b. Injury Symptoms: Banvel causes sugarbeet injury that is visually indistinguishable from phenoxy acetic acid injury (Photo 4). Banvel may have sufficient residual in the soil to reduce emergence and to injure emerging sugarbeet. Emerging seedlings may be twisted, and development of the first true leaves may be inhibited (Photo 5). Banvel residual in soil can cause the "trumpeting" symptom (Photo 6) which also can be caused by early postemergence exposure to phenoxy acetic acid herbicides.
- c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

Photo 4. Petiole epinasty and prostrate leaves from postemergence Banvel. (15KB color photo)

Photo 5. Severely stunted and twisted sugarbeet seedling from Banvel residual in soil. (12KB color photo)

Photo 6. "Celery stalking' or "trumpeting" from Banvel residual in soil. (15KB color photo)

3. Pyridines

- a. Use: Clopyralid (Stinger) for small grains, sugarbeet, corn and grass pastures. Picloram (Tordon) for noncropland, small grains, and grass pastures. Triclopyr for noncropland and grass pasture.
- b. Injury Symptoms: Pyridine injury symptoms are very similar to the phenoxy acetic acids or Banvel. Stinger is registered on sugarbeet but can injure sugarbeet at high rates with a warm, moist environment that favors phytotoxicity. The leaves lay flat and the petioles exhibit epinasty from Stinger just as from phenoxy acetic acids or Banvel (Photo 7). Also, leaves may become more strap-shaped than normal. However, Stinger injury often causes leaves to roll upward from the edges (Photo 8). Leaf rolling is caused to a greater extent by clopyralid than by phenoxy acetic acids or Banvel.
- c. Site of Action: Specific site(s) unknown, believed to have multiple sites of action.

Photo 7. Petiole epinasty and prostrate leaves from postemergence Stinger. (16KB color photo)

Photo 8. Upward curling of leaf margins from postemergence Stinger. (16KB color photo)

II. Amino Acid Synthesis Inhibitors

The amino acid synthesis inhibitors include the herbicide families: sulfonylurea, imidazolinone, 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).

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. (6KB b&w illustration)

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 (one to two 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 families vary greatly in selectivity and may control annual and perennial broadleaf or grass weeds and may be soil or foliar applied. Glyphosate, an amino acid derivative herbicide, is nonselective and the site of uptake is the plant foliage. Glyphosate moves via the phloem to all parts of the plant and is an excellent perennial weed control herbicide and is active on annual weeds as well.

1. Imidazolinones

a. Use:

Imazamethabenz (Assert) for wheat, barley and sunflower. Imazaquin (Scepter) for soybean and noncropland. Imazethapyr (Pursuit) for soybean, dry bean, alfalfa, noncropland and pea.

- b. Injury Symptoms: Same as for sulfonylureas, see next section.
- c. Site of Action: Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

2. Triazolopyrimidine sulfonanilide or sulfonamide

- a. Use: Flumetsulam (Broadstrike) for corn and soybean.
- b. Injury Symptoms: Same as for sulfonylureas, (see next section).
- c. Site of Action: Acetolactate synthase (ALS) enzyme. Also referred to as acetohydroxy acid synthase (AHAS).

3. Sulfonylureas

a. Use:

Chlorimuron (Classic) for soybean. Primisulfuron (Beacon) for corn. Thifensulfuron (Harmony Extra) for small grains. (Pinnacle) for soybean. Triasulfuron (Amber) for small grains. Nicosulfuron (Accent) for corn. Metsulfuron (Ally) for small grains, grass pastures, and CRP. Tribenuron (Express, Harmony Extra) for small grains. Rimsulfuron (Matrix) for potato. Triflusulfuron (Upbeet) for sugarbeet.

b. Injury Symptoms from Soil Residual: Symptoms from imidazolinone, sulfonylurea and sulfonamide herbicides are identical. Not all injured plants will exhibit all symptoms, and symptoms may vary from field to field.

Sugarbeet plants may be stunted and the leaves usually become a bright yellow with first yellowing on young leaves (Photo 9.) An injured plant is on the left and an undamaged plant on the right.) Relatively high levels of herbicide residual in soil may cause the plants to form a rosette rather than a normal sugarbeet plant (Photo 10). The total root and hypocotyl of sugarbeet seedlings may turn brown and shrivel (upper plant, Photo 11) or the root may turn brown and die starting at the point where the root joins the hypocotyl, about 1 to 1.5 inches below the soil surface (lower plant, Photo 11). Plants with injury similar to the upper plant in Photo 11 would probably die due to a non-functional root system, but plants with injury similar to the lower plant often will survive by producing secondary roots from the hypocotyl. However, low moisture in the surface 2 inches of soil can prevent the successful production of secondary roots and the damaged plant would then die.

Photo 9. Yellow and stunted sugarbeet from Classic residual in soil on the left, untreated plant on the right. (14KB color photo)

Photo 10. Stunted and rosetted sugarbeet from Pursuit residual in soil. Knife is 3.5 inches long, picture taken in late July. (14KB color photo)

Photo 11. Root and hypocotyl of the upper plant are brown and shriveled while the root of the lower plant is brown below the point where the root joins the hypocotyl. Symptoms were caused by Pursuit residual in soil. (18KB color photo)

Nearly identical symptoms on roots of seedling sugarbeet also can be caused by dinitroaniline herbicides and *Aphanomyces cochlioides*, a fungal disease. Plants that survive and grow may produce new leaves that are more

strap-shaped than normal (Photo 12).

Photo 12. Sugarbeet plants that survived sulfonylurea herbicide residual in soil and are producing strap-shaped leaves. Leaves did not initiate in pairs since several leaves are all about the same size. (27KB color photo)

c. Injury Symptoms from Postemergence Exposure: Symptoms from imidazolinone and sulfonylurea herbicides are identical. Not all injured plants will exhibit all symptoms and symptoms may vary from field to field.

Plant leaves will become prostrate a few hours after exposure (Photo 13) similar to the effect from phenoxy acetic acids, Banvel or pyridines. Old leaves may remain prostrate for several weeks. However, the petiole epinasty from imidazolinones or sulfonylureas is less than from phenoxy acetic acids, Banvel or pyridines. Yellowing of the youngest leaves begins about four to five days after exposure (Photo 14) and the yellowing intensifies and spreads to the older leaves with time (Photo 15). Severely affected leaves or whole plants may die and turn brown. Petioles may turn black or have black streaks as symptoms worsen. The color contrast between affected and normal plants can become quite evident (Photo 16). The yellow may disappear later in the season as affected plants recover and begin to produce new leaves.

Some affected plants may develop brown rings in the roots within five to seven days after exposure (Photo 17). These rings may still be present at harvest (Photo 18).

Photo 13. Harmony Extra was applied at 0.001 lb/A three days prior to the photo. Leaves are prostrate but the plants have not yellowed. (27KB color photo)

Photo 14. Pursuit was applied at 0.005 lb/A eight days prior to the photo. The plants have started yellowing and the small, young leaves are a bright yellow. (28KB color photo)

Photo 15. Harmony Extra was applied at 0.001 lb/A. The yellowing has spread from the new leaves to the older leaves. One of the older leaves has died and turned brown. Petioles have black streaks near the crown. (29KB color photo)

Photo 16. An accidental application of a sulfonylurea herbicide illustrating the distinctive yellowing of treated plants. (36KB color photo)

Photo 17. Brown rings in roots caused by Harmony Extra foliarly applied six days prior to the photograph. (24KB color photo)

Photo 18. Sugarbeet roots at harvest from Harmony Extra treated plants. Roots have brown rings and abnormal shapes. (20KB color photo)

Plants injured by imidazolinones or sulfonylureas often produce new leaves in clusters rather than in pairs. This can result in more than one crown per root (Photo 19). These plants may be more difficult to defoliate than normal plants. Young seedling exposure to imidazolinone or sulfonylurea herbicides can cause root symptoms similar to those from soil residual (Photo 11).

Photo 19. Pinnacle-treated sugarbeet at harvest. A normal sugar-beet has one crown of leaves but this plant initiated several crown areas where leaves were produced in clusters. (29KB color photo)

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

4. Amino Acid Derivatives

- a. Use: **Glyphosate (Several trade names)** nonselective weed control before crop emergence, for spot treatments in some crops, in pasture and in noncropland.
- b. Injury Symptoms: Sugarbeet injury from glyphosate is quite similar to injury from imidazolinones or sulfonylureas. However, the yellowing from exposure to glyphosate starts with the older leaves and moves toward the younger leaves (Photo 20) while injury from imidazolinones or sulfonylureas starts with the younger leaves and moves toward the older leaves (Photo 15). Glyphosate can cause brown coloring in the roots (Photo 21) similar to the imidazolinones or sulfonylureas.
- c. Site of Action: 5-enolpyruvyl-shikimate-3 phosphate synthase (EPSP synthase) enzyme.

Photo 20. Glyphosate-treated sugarbeet with yellowed older leaves and green new leaves. Leaf yellowing from glyphosate progresses from old leaves to new leaves. (28KB color photo)

III. Lipid Inhibitors

The lipid inhibitors include the aryloxyphenoxypropionate and cyclohexanedione herbicide families: 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 bio-synthesis (Figure 1). Broadleaf plants are tolerant to these herbicide families, but almost all perennial and annual grasses are susceptible. Injury symptoms are slow to develop (seven to 10 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 Plus) for soybean and alfalfa.
 (Poast) for soybean, alfalfa, sugarbeet, flax, sunflower, lentil, pea and potato.
 Clethodim (Select) for soybean.
- b. Injury Symptoms: Sugarbeet is not injured by cyclohexanediones even at high rates.
- c. Site of Action: Acetyl-CoA carboxylase enzyme.

2. Aryloxyphenoxypropionates

a. Use:

Diclofop (Hoelon) for small grain. Fluazifop (Fusilade 2000) for soybean. Fenoxaprop (Whip, Option II) for soybean and (Dakota, Tiller, Cheyenne) on small grain. Quizalofop (Assure II) for soybean.

- b. Injury Symptoms: Sugarbeet is not injured by aryloxyphenoxypropionates used alone but would be injured by mixtures with other herbicides such as in Dakota, Tiller or Cheyenne.
- c. Site of Action: Acetyl-CoA carboxylase enzyme.

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