Herbicide Mode of Action

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Herbicides are chemicals that inhibit or interrupt normal plant growth and development. They are used widely in agriculture, industry, and urban areas for weed management. Herbicides can provide cost-effective weed control with a minimum of labor. However, improper herbicide use can injure crops, damage the environment, and pose a threat to the applicator and others exposed to the chemical. Herbicide mode of action is how herbicides work. Understanding how herbicides work provides insight into how to use the chemicals and helps diagnose performance problems and related injury symptoms.

The best source of information for herbicide use is the herbicide label. Always apply herbicides according to label directions. Publications such as the K-State Research and Extension Report of Progress, *Chemical Weed Control for Field Crops, Pastures, Rangeland, and Noncropland*, also provide information on available herbicide options and application guidelines. The herbicide label, however, is a legal document, and an applicator is responsible for applying the herbicide according to label directions.

Herbicides kill plants in different ways. A herbicide must meet several requirements to be effective. It must come in contact with the target weed, be absorbed by the weed, move to the site of action in the weed, and accumulate sufficient levels at the site of action to kill or suppress the target plant. Weed control is unsatisfactory unless these requirements are met.

All herbicide interactions with a plant, from application to final effect, are considered the mode of action. The herbicide mode of action involves absorption into the plant, translocation or movement in the plant, metabolism of the herbicide, and the physiological plant response. Herbicide site of action refers to the specific process in the plant that the herbicide disrupts to interfere with plant growth and development.

Herbicides may be classified according to selectivity (nonselective, grass control, broadleaf control, etc.), time of application (preplant incorporated, preemergence, or postemergence), translocation in the plant (contact or systemic), persistence, or site of action.

Herbicide Selectivity

The herbicide's ability to kill certain plants without injuring others is called selectivity. Herbicides that kill or suppress the growth of most plant species are relatively nonselective. Nonselective herbicide use is limited to situations where control of all plant species is desired, or by directing the herbicide on the target weed and away from desirable plants. Glyphosate and paraquat historically have been considered nonselective herbicides. However, glyphosate is a highly selective herbicide when used in conjunction with crops that have been genetically engineered with resistance to glyphosate.

Most herbicides used in crop production are selective. Herbicide selectivity is relative and depends on several factors, including environment, herbicide application rate, application timing, and application technique. Even tolerant plant species may be susceptible to a herbicide if the application rate is high enough. Herbicide selectivity may be based on herbicide placement, differential spray retention, absorption, translocation, metabolism, or an altered site of action.

Herbicide Placement

Herbicide placement can be critical to effectiveness and selectivity. Most small weed seeds germinate and emerge from the top 1/2 inch of soil. Herbicides applied and positioned near the soil surface will be most available for absorption by shallow-germinating weed seeds. However, larger seeded weeds that emerge from deeper in the soil may not be controlled very well by a preemergence herbicide unless it is incorporated or moved deep enough into the soil by water movement. Selectivity may be achieved by seeding the crop below the herbicide-treated zone, especially if the herbicide is root absorbed and relatively immobile in the soil.

Spray Retention

Greater spray retention by a plant is likely to result in more herbicide absorption. Spray retention depends on the properties of the spray solution and the target plant. Leaf waxiness, pubescence (hairiness), and orientation are among the characteristics that affect spray retention. Waxy leaf surfaces repel water-based spray solutions, allowing spray droplets to run off more easily than on less waxy leaves. Sparse leaf pubescence or hairs may help retain spray droplets, but dense pubescence can hold spray droplets above the leaf surface and reduce spray contact with the leaf (Figure 1).

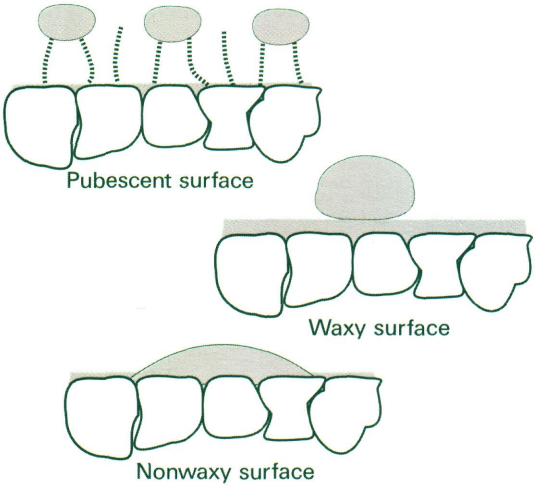

Figure 1. Spray droplet spread on a leaf surface as influenced by leaf pubescence and waxiness.

Growth Habit

Grass plants tend to be more difficult to wet than broadleaf plants because grasses often have narrow, waxy leaves with upright orientation. The grass leaf presents a small target, leaving a good chance the spray droplet will roll off the leaf upon contact. Broadleaf plants may be easier to wet because they present a large target with some pubescence and horizontal orientation (Figure 2).

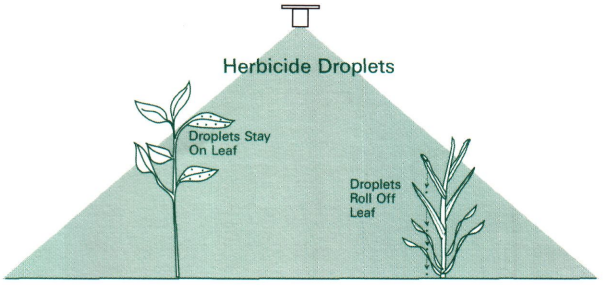

Figure 2. Spray droplet retention on grass and broadleaf leaves due to leaf orientation.

Spray Adjuvants

Spray adjuvants or additives often improve spray retention and absorption by reducing the surface tension of the spray solution, allowing the spray droplet to spread more evenly over the leaf surface (Figure 3). Herbicide absorption may be further enhanced by interacting with the waxy cuticular layer on the leaf surface.

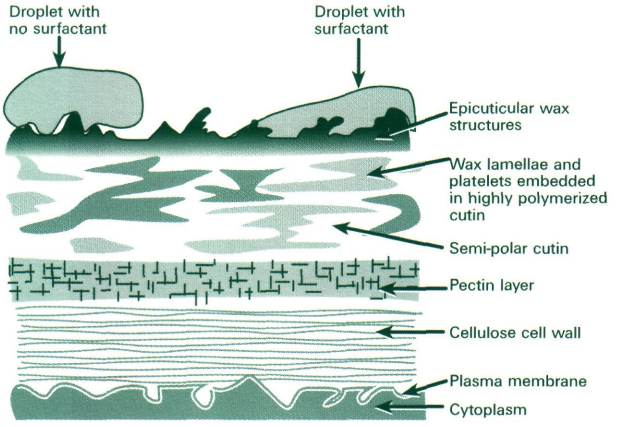

Figure 3. Leaf surface composition and the influence of surfactants on droplet spread over the leaf surface (adapted from Hull, Davis, and Stolzenberg).

Table 1. Scepter selectivity due to differential metabolism in different plant species (Shaner and Robson, 1985, Weed Science 33:469-471).

Plant species	Scepter remaining in plants after 3 days	Scepter half-life in plants (days)	Plant response
Common cocklebur	99%	30	Very Susceptible
Soybean	38%	3	Tolerant
Velvetleaf	89%	12	Susceptible

Spray additives can increase weed control, but potentially can reduce selectivity by increasing the spray retention and herbicide absorption by the crop more than by the weed. Spray additives should be used only if recommended on the herbicide label. Refer to K-State Research and Extension publication *Spray Adjuvants with Herbicides,* MF-1043, for more information on the function and use of spray additives.

Herbicide Metabolism

Metabolism is one of the most important ways a plant can escape the toxic effects of a herbicide. Herbicide-tolerant plants often have the ability to metabolize or break down the chemical to nonactive compounds before it can build up to toxic levels at the site of action. Susceptible plants are unable to detoxify herbicides. Selectivity of many herbicides is based on differing rates of metabolism. Table 1 illustrates differential metabolism and tolerance of Scepter among soybeans, velvetleaf, and common cocklebur.

Altered Site of Action

An altered site of action can result in dramatic resistance to a herbicide. An altered site of action refers to genetically different plant biotypes that have a structurally altered site of action that prevents herbicide binding and activity. An altered site of action can be visualized using the lock-and-key concept illustrated in Figure 4. Altered site of action has been the basis for many herbicide-resistant weed problems. Kochia resistance to atrazine or Glean is an example of herbicide resistance due to an altered site of action. A small percentage of the original weed population is genetically different and contains the resistant trait. Repeated use of the herbicide or herbicides with the same site of action results in removal of susceptible biotypes, while resistant biotypes increase until the weed population is no longer controlled effectively with that group of herbicides. Weeds that are resistant to a specific herbicide often are also resistant to other herbicides with the same site of action. Refer to K-State Research and Extension publication *Questions & Answers on Managing Herbicide-Resistant Weeds,* MF-926, for more information.

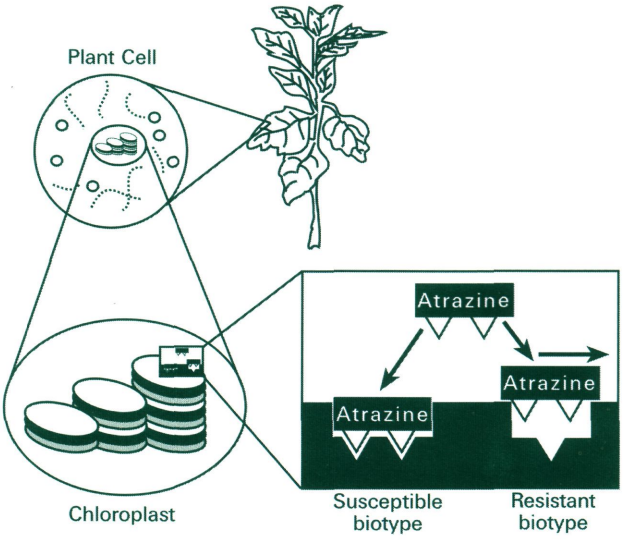

Figure 4. Site exclusion type of herbicide resistance. Atrazine is ineffective on resistant biotype because a conformational change in the chloroplast prevents it from binding at the site of action (adapted from Gonsolus).

Herbicide Translocation in Plants

Systemic herbicides are translocated in plants, while contact herbicides are not translocated. To be effective, contact herbicides must be applied to the site of action. Most foliar-applied contact herbicides work by disrupting cell membranes. Thorough spray coverage of a plant is essential with foliar-applied contact herbicides to kill the entire plant.

Contact herbicides generally are ineffective for long-term perennial weed control. Contact herbicides damage the top growth that the spray solution contacts, but the underground portion of perennial plants remains unaffected and can rapidly initiate new growth.

Contact herbicides often are more effective on broadleaves than on grasses. The growing point of young grasses is located in the crown region of the plant, which is at or below the soil surface, and thus, difficult to contact with the spray. In contrast, the

growing point on young broadleaf plants is exposed to the spray treatment. Thus, paraquat may not kill all the growing points of a tillered grass plant, and regrowth can occur.

Systemic herbicides can be translocated to other parts of the plant either in the xylem or the phloem (Figure 5). The xylem is nonliving tissue through which water and nutrients move from the roots to the shoots and leaves of plants. Translocation in the xylem is only from the roots to the leaves. Phloem is a living, conducting system in which materials can move both upward and downward. The phloem transports the food that is produced in the leaves to the roots and to areas of new growth (Figure 5).

Herbicides can be translocated in the xylem, the phloem, or both. Translocation depends on the chemical and the plant species. Herbicides translocated only in the xylem are most effective as soil-applied or early postemergence treatments because translocation is only upward. Atrazine is a good example of a herbicide that is translocated only in the xylem. Phloem translocated herbicides that move downward and suppress root and rhizome growth, as well as top growth, provide the best perennial weed control. Tordon, 2,4-D, Banvel, and Roundup are examples of systemic herbicides that will translocate in the phloem and provide good, long-term control of certain perennial weeds.

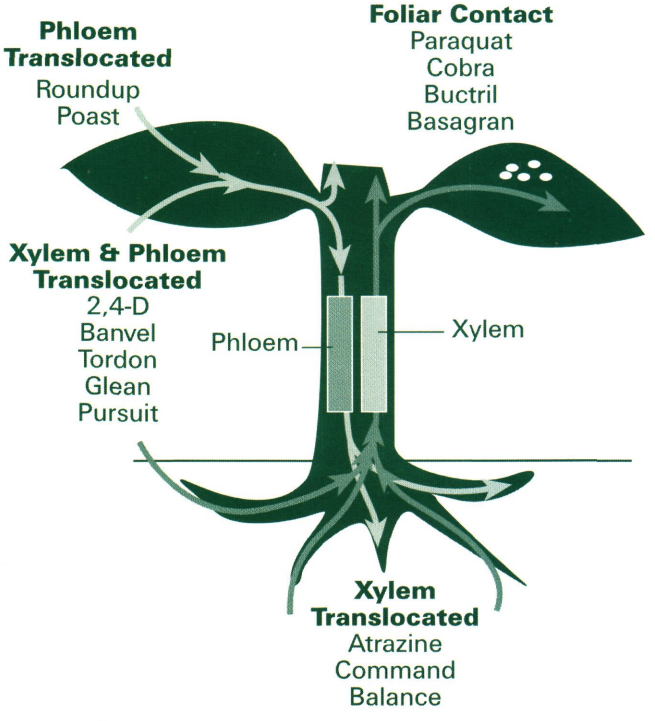

Figure 5. Herbicide translocation in plants.

Factors Affecting Herbicide Activity

Factors influencing herbicide activity include application rate, application technique, plant maturity, and environmental conditions. In addition, soil characteristics can affect soil-active herbicides.

Moisture and temperature are environmental factors that influence activity of soil-applied herbicides. Precipitation is essential to move surface-applied or preemergence herbicides into the soil and activate them. Mechanically incorporated herbicides tend to provide more consistent weed control than surface-applied herbicides because the herbicide is in place, and adequate moisture usually is present in the soil to activate the chemical. However, incorporation too deep may dilute the herbicide so weed control is poor. Improperly adjusted equipment, or incorporation when soils are too wet, may result in streaking and poor weed control.

Soil moisture is important because it influences herbicide adsorption to soils. Therefore, the herbicide is unavailable for plant uptake. Adsorption occurs when herbicide molecules adhere to soil particles and organic matter. While adsorbed, herbicide molecules are unavailable for absorption by plants. Water molecules compete with herbicide molecules for adsorption sites on soil particles and organic matter. Therefore, herbicide adsorption is highest under dry soil conditions, and lowest in moist soils. Consequently, weed control is generally best with moist soil conditions because more herbicide is available for plant uptake in the soil solution or gaseous phase.

Temperature affects the activity of soil-applied herbicides primarily because of its influence on the rate of seed germination, emergence, and growth. Seedling plants tend to be more susceptible to soil-applied herbicides under cool conditions than under warm temperatures because plant emergence is delayed and metabolism is slowed. On the other hand, extremely high temperatures sometimes increase crop injury simply by placing the plant under multiple stresses.

Soil characteristics affecting herbicide activity are texture, organic matter, and pH. Herbicide adsorption is greater in fine-textured soils high in organic matter than in coarse-textured soils low in organic matter. Thus, a lower proportion of herbicide is available in the fine-textured soils, so a higher herbicide application rate is required to provide the same level of weed control as in a coarse-textured soil. At the same time, the chance of

crop injury is greater on coarse-textured soils low in organic matter because a higher proportion of the applied herbicide is available for plant uptake. Soil-applied herbicide rates usually need to be adjusted according to soil texture and organic matter content.

Soil pH influences the availability and persistence of certain herbicides in the soil. Soil pH can alter the ionic nature of the herbicide molecule, which influences adsorption, solubility, and rate of herbicide breakdown. The triazine herbicides (atrazine, Sencor, and simazine) and some of the sulfonylurea herbicides (Amber, Finesse, Peak, Exceed, Oust, and Classic) are more active and more persistent in high pH soils (> 7.0) than in low pH soils. Refer to K-State Research and Extension publication *Residual Herbicides, Degradation, and Recropping Intervals,* C-707, for more information.

Environmental conditions can have a two-fold effect on the performance of postemergence herbicides. Higher humidity and favorable temperatures generally result in greater herbicide absorption and activity in plants.

Environment also influences herbicide efficacy by affecting plant growth. Plants are generally most susceptible to postemergence herbicides when actively growing. Extreme environmental conditions that slow plant growth and thicken leaf cuticles often increase plant tolerance to a herbicide. Crop injury from a herbicide, however, can increase during poor growing conditions because of slower metabolism and detoxification of the herbicide. Thus, if crop tolerance is based on the ability of the crop to rapidly metabolize the herbicide, the potential for crop injury may increase and weed control decrease if a herbicide is applied when plants are not growing actively. For this reason, most herbicide labels caution against application during extreme environmental conditions.

Annual plants are usually more susceptible to herbicides when they are small than when they are mature. As they mature, plants develop thicker wax layers on leaf surfaces, reducing herbicide absorption. In addition, it is harder to achieve thorough spray coverage on large plants than on small plants.

Established perennial weeds tend to be more susceptible to herbicides if applied during the early flowering stage of growth or to actively growing plants in the fall, probably because application at these times results in the greatest translocation of the herbicide to the roots. However, true seedlings are much easier to control than established perennial weeds.

Herbicide Sites of Action

Herbicides can work at various sites in plants. They generally interfere with a process essential for normal plant growth and development. Herbicides can be classified by site of action based on how they work and the injury symptoms they cause. The Weed Science Society of America (WSSA) has developed a numbered classification system based on the herbicide site of action. Knowledge of herbicide sites of action can allow proper selection and rotation of herbicides to reduce the risk of developing herbicide resistant weeds. Classification of herbicides by site of action and the WSSA classification number (in parenthesis) are described below.

Growth Regulators (4)

Phenoxy	2,4-D, 2,4-DB (Butyrac) 2,4-DP, MCPA, MCPP
Benzoic acid	dicamba (Banvel, Clarity, Distinct)
Carboxylic Acid	picloram (Tordon) clopyralid (Stinger) fluroxypyr (Starane) triclopyr (Garlon, Remedy, Crossbow)
Quinoline	quinclorac (Paramount)

Growth regulator herbicides are used primarily for controlling broadleaf weeds in grass crops and pastures and include some of the more effective chemicals for perennial broadleaf weed and brush control. Most growth regulator herbicides are readily absorbed through both roots and foliage and are translocated in both the xylem and phloem (Figure 5). Translocation of foliar-applied treatments, however, is more restricted in grasses than in susceptible broadleaves.

These herbicides are called growth regulators because they mimic natural growth hormones, and thus, upset the natural hormone balance in plants. Growth hormones regulate cell elongation, protein synthesis, and cell division. The killing action of growth-regulating chemicals is not caused by any single factor, but rather by the disruption of several growth processes in susceptible plants.

Injury symptoms on susceptible plants treated with growth regulator herbicides include growth and reproduction abnormalities, especially on new

Photos 1 and 2. Growth regulator herbicides often cause abnormal leaf growth and development, such as the cupping symptom on soybean leaves from dicamba injury (top) and the puckering and parallel venation in the soybeans from 2,4-D (bottom).

Photo 3. Grasses treated with growth regulator herbicides sometimes exhibit leaf rolling or "onion-leafing" similar in appearance to drought stress. Injury can be exagerated by tank mixes with other herbicides or the addition of adjuvants.

growth. Broadleaf species exhibit stem and petiole twisting (epinasty), leaf malformations (parallel venation, crinkling, leaf strapping, and cupping), (Photos 1 and 2) stem callus formation, and stunted root growth. Grass plants exhibit rolled leaves (onion leafing) (Photo 3), fused brace roots (Photo 4), leaning stems (Photo 5), and stalk brittleness (Photo 6). Growth regulator herbicides may affect reproduction, resulting in sterile or multiple florets (Photos 7 and 8), and nonviable seed production.

Environmental and use considerations. Growth regulator herbicides can cause serious drift injury to susceptible plants (i.e. tomatoes, grapes, cotton, sunflowers, soybeans, cucumbers). The phenoxy herbicides may be formulated as esters or amines. The esters are volatile and can cause vapor drift damage, while the amines are relatively nonvolatile. Banvel, Clarity, and Distinct also are subject to

Photo 4. Growth regulator herbicides can interfere with normal root growth as exhibited by the brace roots on this corn plant that was treated with 2,4-D.

Photos 5 and 6. Growth regulator herbicides such as 2,4-D and dicamba can cause the stems of grasses to lean over and become weakened after application (top), after which the stems may exhibit "goosenecking" as plants try to grow upright (bottom). May also cause complete stem breakage, called "green snap," from which plants do not recover.

Photo 7. Misapplied growth regulator herbicides can cause sterility, twisted awns, and head trapping in small grains.

Photo 8. Late applications of plant growth regulator herbicides such as dicamba and 2,4-D can interfere with pollination and seed production, sometimes called headblasting in sorghum. Cold night temperatures during pollination can also cause poor grain fill.

vapor drift. Vapor drift increases as temperatures increase, and may occur as late as several days after application.

The phenoxies are relatively short-lived in the environment and have small pollution potential. Tordon is water soluble and persistent in the soil. Consequently, Tordon has a high leaching potential and should not be used on coarse-textured soils with a shallow water table, where groundwater contamination is most likely to occur.

Seedling Growth Inhibitors

The seedling growth inhibitors work during germination and emergence and include three groups: 1) the seedling shoot inhibitors (carbamothioates), 2) the seedling shoot and root inhibitors (acetamides), and 3) the microtuble assembly inhibitors (dinitroanilines).

Seedling Shoot Inhibitors (8):

Carbamothioates	EPTC (Eradicane, Eptam) butylate (Sutan+) triallate (Far-Go)

Carbamothioate herbicides are soil-incorporated for control of annual grasses and some broadleaf weeds. All are volatile and need to be incorporated immediately after application to avoid excessive vapor loss. Vapor loss of the carbamothioate herbicides is less when applied to dry soils than when applied to moist soils. The carbamothioates are absorbed from the soil solution or vapor phase through both roots and emerging shoots, but are translocated only in the xylem. The primary site of absorption and action is the emerging shoot and growing point. The mechanism of action of these herbicides is not well understood, but they seem to interfere with normal cell development in the newly developing shoot.

Corn tolerance to the carbamothioate herbicides increases with use of dichlormid safener (formulated in Eradicane and Sutan+), which increases metabolism of the chemicals to nontoxic substances. Dichlormid is unique because it can be applied with the spray formulation at low rates and selectively protects only corn against herbicide injury.

Repeated use of the carbamothioate herbicides on the same field results in a buildup of microbes that break down the herbicides, decreasing their residual life and period of weed control. This phenomenon is known as "enhanced degradation" or soil conditioning. Repeated use of one carbamothioate herbicide also conditions the soil for enhanced degradation of the other carbamothioates. The best way to avoid enhanced degradation of herbicides is to rotate to a different class of herbicides and avoid application of carbamothioates in successive years.

Injury symptoms on grass plants include failure of the shoot to emerge from the coleoptile or whorl of the plant, giving the plant a buggy-whip appearance. Susceptible grass seedlings often fail to emerge from the soil. Injury symptoms on broadleaf plants include enlarged cotyledons, restricted growth of the true leaves, and a dark green color, a symptom sometimes referred to as bud seal. The roots become short, thick, brittle, and club-shaped.

Seedling Shoot and Root Inhibitors (15):

Acetamide	alachlor (Lasso, Micro-Tech, Partner) *S*-metolachlor (Dual MAGNUM) propachlor (Ramrod) dimethenamid (Frontier) acetochlor (Surpass, Topnotch, Harness, Degree) flufenacet (Axiom, Define) *P*-dimethenamid (Outlook)

Acetamide herbicides are used preemergence or with shallow soil incorporation to control annual grasses and some broadleaf weeds in a variety of crops. The acetamides do not control emerged plants. The primary site of absorption and action of these herbicides on broadleaf species is the roots, while the primary site of absorption and action on

grass species is the emerging shoot. The acetamides are not readily translocated in the plant, so herbicide placement and availability are important. As with the carbamothioates, the mechanism of action of the acetamides has not been well defined, but appears similar to the carbamothioates. These herbicides affect various biochemical processes in the plant and interfere with normal cell development.

Photo 9. Acetamide herbicides can cause emergence problems and distorted shoots of grasses, such as this unsafened sorghum that was treated with Dual. Only Concep treated sorghum seed should be planted if acetamide herbicides will be used (except Ramrod).

Lasso, Micro-Tech, Partner, Dual MAGNUM, Frontier, and Outlook may be used in sorghum if the seed is treated with Concep seed protectant. The seed protectant increases sorghum tolerance to the acetamide herbicides by increasing metabolism of the herbicide to inactive compounds.

Injury symptoms caused by the acetamides are similar to those caused by the thiocarbamates. The new shoots fail to emerge from the coleoptile and whorl of the shoot of grass species (Photo 9). Susceptible germinating grasses often fail to emerge from the soil. Injury symptoms on broadleaf species include general stunting and a drawstring effect around the margins of the true leaves (Photo10).

Microtubule Assembly Inhibitors (3):

Dinitroaniline	trifluralin (Treflan) pendimethalin (Prowl, Pendimax) ethalfluralin (Sonalan) benefin (Balan)

Dinitroaniline herbicides are generally applied preplant incorporated to control annual grasses and some broadleaf weeds in many crops. Treflan, Sonalan, and Balan need to be incorporated to avoid photodecomposition and volatility losses. Prowl is less volatile than the other dinitroaniline herbicides and can be applied preemergence, but generally provides better weed control when soil-incorporated. The dinitroaniline herbicides are absorbed by both roots and shoots of emerging seedlings, but are not readily translocated (Figure 5). The emerging shoot is the primary site of absorption and action on grass species. These herbicides are mitotic poisons that inhibit cell division. Thus, the meristematic regions, such as the growing points of stems and roots, are most affected. Selectivity may be based on metabolism, as well as herbicide placement and type of emergence of the grass species.

Injury symptoms on grass species include short, swollen coleoptiles. Injured broadleaf plants often have swollen hypocotyls. Preemergence pendimethalin sometimes causes callus formation

Photo 10. Acetamide herbicides sometimes cause minor stunting and distorted leaves such as the heart-shaped leaf on this soybean plant, which resulted from Lasso injury following cold, wet weather during emergence.

Photo 11. Preemergence pendimethalin can cause callus formation and brittle stems on soybeans, resulting in breakage and lodging.

Photo 12. DNA herbicides like Treflan and Prowl can cause poor root development and short, stubby roots.

and brittle stems near the soil surface, which may break over during the growing season (Photo 11). Both grasses and broadleaves may have short, stubby secondary roots (Photo 12). As a consequence, the plants may be stunted and exhibit nutrient deficiency or drought symptoms because of the poorly developed root system.

Environmental and use considerations. The carbamothioates and dinitroanilines are characterized by low solubility in water and high adsorption to soils. Thus, they are not readily leached or moved in water. The acetamides are more soluble and less adsorptive, but less persistent in the soil.

Photosynthetic Inhibitors

Photosystem II, Site A (5):

Triazine	atrazine simazine (Princep) ametryn (Evik) prometon (Pramitol)
Triazinone	metribuzin (Sencor) hexazinone (Velpar)
Uracil	terbacil (Sinbar) bromacil (Hyvar)

Photosystem II, Site B (7):

Phenylurea	linuron (Lorox, Linex) diuron (Karmex, Diurex) tebuthiuron (Spike)

Photosystem II, Site C (6):

Benzothiadiazole	bentazon (Basagran)
Nitrile	bromoxynil (Buctril, Moxy)
Phenyl-pyridazine	pyridate (Tough)

Photosynthetic inhibitor herbicides control many broadleaf and some grass weeds. All of these herbicides work by disrupting photosynthesis, but there are three different binding sites. Binding site A includes the triazines, triazinones, and uracils, binding site B includes the phenylureas, and binding site C includes Basagran, bromoxynil and Tough.

The triazines, triazinones, uracils, and phenylureas are soil-applied or early postemergence herbicides in crops and noncropland sites. These herbicides are absorbed by both shoots and roots, but are translocated only in the xylem (Figure 5).

Basagran, Tough, and bromoxynil are used primarily as early postemergence treatments. They are contact herbicides that are not translocated in the plant (Figure 5). Thorough spray coverage of the foliage is essential for good weed control with these herbicides.

These herbicides block photosynthesis, the food production process in plants. Plants are not affected by the herbicide until after they emerge and begin photosynthesis. Even though photosynthesis is inhibited, susceptible plants do not die simply from starvation. Herbicide injury symptoms appear too quickly and are not typical of starvation. Instead, susceptible plants treated with a photosynthetic inhibitor die from a buildup of highly reactive molecules that destroy cell membranes.

The selective action of triazine herbicides is primarily determined by differential metabolism. Plant species such as corn and sorghum possess the glutathione-S-transferase enzyme and can selectively metabolize triazine herbicides into nontoxic substances. Crop and weed selectivity to urea herbicides, such as Lorox, is due primarily to herbicide placement rather than metabolism or differential physiological tolerance of plant species.

Injury symptoms from soil-applied treatments will not appear until after photosynthesis begins. Susceptible broadleaf plants will exhibit interveinal chlorosis and necrosis beginning around the leaf margins and progressing toward the center of the leaves (Photo 13). Susceptible grasses will become chlorotic and necrotic beginning at the leaf tips and

Photo 13. Photosynthetic inhibitors can cause chlorosis and necrosis around the margins of the oldest leaves of susceptible broadleaves. This injury could be from atrazine carryover, metribuzin damage, or a combination of the two.

Photos 14 and 15. Atrazine can carry over and cause damage to susceptible crops. Carryover injury is most likely on high pH soils or in areas with higher application rates such as turn row overlaps (top). Susceptible plants will emerge, followed by leaf yellowing and dieback beginning at the tips (bottom).

Photo 16. ALS herbicide injury generally appears as chlorosis and general stunting of the growing point, followed by gradual death of the plants. The tansy mustard in this wheat was treated with Glean.

progressing toward the base of the leaves (Photos 14 and 15). Injury symptoms from foliar applications will appear as leaf burn as cell membranes are destroyed. Leaf burn symptoms generally occur most rapidly with hot, humid conditions.

Environmental and use considerations. Basagran, Tough, and bromoxynil are foliar-applied and relatively short-lived in the environment. They do not pose a serious environmental threat. The other photosynthetic inhibitor herbicides are primarily soil-applied and have fairly long persistence in the soil. These herbicides may contaminate surface water in regions with fine-textured soils and groundwater in regions with coarse-textured soils and shallow water tables.

Amino Acid Synthesis Inhibitors

Acetolactate Synthase (ALS) inhibitors (2):

Sulfonylurea	chlorsulfuron (Glean) metsulfuron (Ally, Escort) tribenuron (Express) triasulfuron (Amber) chlorimuron (Classic) thifensulfuron (Pinnacle) sulfometuron (Oust) primisulfuron (Beacon) nicosulfuron (Accent) halosulfuron (Permit) prosulfuron (Peak) rimsulfuron (component of Basis) sulfosulfuron (Maverick Pro) pyrithiobac (Staple)
Imidazolinone	imazaquin (Scepter) imazethapyr (Pursuit) imazapic (Plateau) imazapyr (Contain) imazamox (Raptor)
Triazolopyrimidine	flumetsulam (Python) cloransulam (FirstRate)

The acetolactate synthase (ALS) inhibiting herbicides have a broad spectrum of selectivity and are used at low rates as soil-applied or postemergence treatments in a variety of crops. These herbicides inhibit the activity of the ALS enzyme, which is involved in the synthesis of the branch chain amino acids (leucine, isoleucine, and valine). Amino acids are essential building blocks in

Photos 17 and 18. Some ALS herbicides can carry over and damage the following crop. These soybeans were damaged by Peak carryover, resulting in yellow, stunted plants (top). Planting ALS resistant or tolerant crops can help alleviate the potential for carryover, as with the STS soybeans (sulfonylurea tolerant) planted in the background of this picture (bottom).

Photo 19. ALS herbicides such as Basis, Exceed, Spirit, Accent, and Beacon can sometimes cause a chlorotic band, crinkled leaves, and bending of new corn leaves coming out of the whorl, especially with cold conditions following application or when applied in conjunction with certain insecticides.

proteins and are required for production of new cells. ALS herbicides are readily absorbed by both roots and foliage and translocated in both the xylem and phloem to the site of action at the growing points (Figure 5). Selectivity is based on differential metabolism and site exclusion. Kochia, Russian thistle, cocklebur, sunflower, shattercane, and pigweed populations resistant to ALS herbicides have developed in some areas of Kansas where these herbicides have been used frequently.

Injury symptoms caused by ALS inhibiting herbicides are not apparent until several days after treatment, although susceptible plants stop growing almost immediately. Affected plants can exhibit

Photo 20. ALS herbicides sometimes cause a purple or red venation of leaves as evident on the underside of this soybean leaf that was treated with Classic herbicide.

stunting, interveinal chlorosis (Photos 16, 17, 18), chlorotic banding on grass leaves (Photo 19), red leaf venation (Photo 20), purpling, root pruning (Photo 21), and gradual death. The risk of crop injury from the sulfonylurea and triazolopyrimidine herbicides is more likely on high than low pH soils (Photo 22).

Environmental and use considerations. These herbicides have exceptionally low mammalian toxicity and have minimal environmental concerns because of the low use rates. Herbicide drift and spray contamination, however, are a concern because susceptible crops are very sensitive to these chemicals. Many of the ALS inhibiting herbicides can carry over in the soil and injure subsequent crops. Carryover of the sulfonylurea herbicides is much greater in high pH soils than low pH soils, while carryover of the imidazolinone and triazolopyrimidine herbicides tends to be more likely in soils with low pH.

Photo 21. ALS herbicide carryover can cause a proliferation of secondary roots or "bottle brushing" on susceptible crops. The roots do not function normally and shoots may be stunted and show nutrient deficiency symptoms.

Photo 22. ALS herbicide injury is sometimes confounded by iron deficiencies on high pH soils resulting in interveinal chlorosis as evident on this grain sorghum that was treated preemergence with Peak herbicide.

Enolpyruvyl-shikimate-phosphate (EPSP) Synthase inhibitors (9):

Amino acid derivative	glyphosate (Roundup, Glyfos, Glyphomax, Acquire, Credit, Touchdown)

The EPSP inhibitor herbicides are readily absorbed through plant foliage and translocated in the phloem to the growing points (Figure 5). These herbicides inhibit the EPSP enzyme, which is involved in the synthesis of the aromatic amino acids (tyrosine, tryptophan, and phenylalanine). Glyphosate is a relatively nonselective postemergence herbicide that is inactive in the soil because of adsorption. Glyphosate resistant crops with an alternative EPSP enzyme have been developed through genetic engineering.

Photo 23. The EPSP inhibitors, such as glyphosate, are systemic herbicides that affect the growing points and cause a gradual discoloration and death of plants. The glyphosate drift to sorghum plants in this picture is causing different degrees of injury, including chlorotic banding of the leaves in the whorl on the center plant and purple and brown plants on either side.

Injury symptoms are not apparent until 3 to 5 days after treatment and include stunting, foliage discoloration, and slow plant death (Photo 23). Grasses exposed to a sublethal dose of EPSP inhibitors may exhibit a chlorotic band across the leaves in the whorl of the plant.

Environmental and use considerations. The EPSP inhibitor herbicides have exceptionally low mammalian toxicity and have minimal pollution concerns because of high adsorption to soil colloids. Herbicide drift and spray contamination, however, are a concern because of the sensitivity of susceptible crops to these chemicals.

Nitrogen Metabolism Inhibitors

Glutamine Synthetase Inhibitors (10):

Phosphorylated amino acid	glufosinate (Liberty)

Liberty is a broad-spectrum postemergence herbicide that has no soil activity. Liberty resistant crops have been developed with genetic engineering. Liberty inhibits the activity of the glutamine synthetase enzyme that is necessary for the plant to convert ammonia into other nitrogen compounds. Consequently, ammonia accumulates and glutamine levels decrease. Plant damage probably occurs due to the combined effects of ammonia toxicity and deficiency of amino acids required for other metabolic processes. Liberty has limited translocation, so thorough spray coverage of small weeds generally provides the best performance.

Injury symptoms appear as foliar burn within several hours following application. Large weeds will often initiate regrowth from axillary buds.

Environmental and use considerations. Liberty has low mammalian toxicity and minimal pollution concerns because of high adsorption to soil colloids.

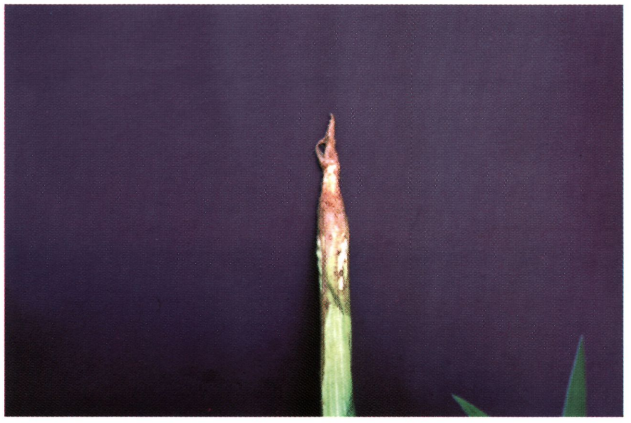

Photo 24. Lipid synthesis inhibitors such as Select, Poast, Fusion, and Assure are systemic herbicides that interfere with the production of new cells at the base of grass leaves. Consequently, about 1 week after treatment the new leaves can be pulled out of the whorl and the leaf tissue at the base of the leaves appears rotten.

Lipid Synthesis Inhibitors

Acetyl-CoA Carboxylase (ACCase) Inhibitors: (1)

Aryloxyphenoxypropionate	diclofop (Hoelon) fluazifop (Fusilade) fenoxaprop (Option) quizalofop (Assure II)
Cyclohexanedione	sethoxydim (Poast, Poast Plus) clethodim (Select)

Lipid synthesis inhibitor herbicides primarily are used postemergence for grass control in broadleaf crops. They have a high degree of selectivity with little or no broadleaf activity. These herbicides are absorbed through the foliage and translocated in the phloem to the meristematic regions (Figure 5). The postemergence grass control herbicides halt meristematic activity by inhibiting the acetyl-CoA carboxylase (ACCase) enzyme that is involved in the synthesis of lipids and fatty acids. Lipids are essential components of cell membranes, and without them, new cells cannot be produced.

Application of the postemergence grass herbicides tank-mixed with a broadleaf herbicide often results in reduced grass control, a response called antagonism. The antagonism can be overcome by applying the separate herbicides several days apart, or by increasing the rate of the grass control herbicide in a tank mix.

Injury symptoms caused by the lipid synthesis inhibitors are not evident until several days after treatment, although the plants cease growing soon after herbicide application. Fully developed leaves of treated grass plants may still look healthy for several days after treatment, but new leaves in the whorl of the plant will pull out easily, exposing decayed tissue at the base of the leaves (Photo 24). The plants will gradually turn purple, brown, and die, but older leaves may stay green for a long time. Drift of sublethal rates of lipid synthesis inhibitors to susceptible grasses can cause a chlorotic band across the leaves in the whorl, damage the stems, or kill the main shoots, depending on the dosage and stage of growth of the plant (Photos 25, 26).

Environmental and use considerations. The selective postemergence grass control herbicides are foliar applied, short-lived in the soil, have a low water solubility, and are used at relatively low rates. Thus, they have a low leaching potential and do not pose a serious threat to the environment.

Photo 25. Sublethal doses of lipid synthesis inhibitor herbicides can cause a chlorotic band across the leaves of grasses as they emerge from the whorl of the plant.

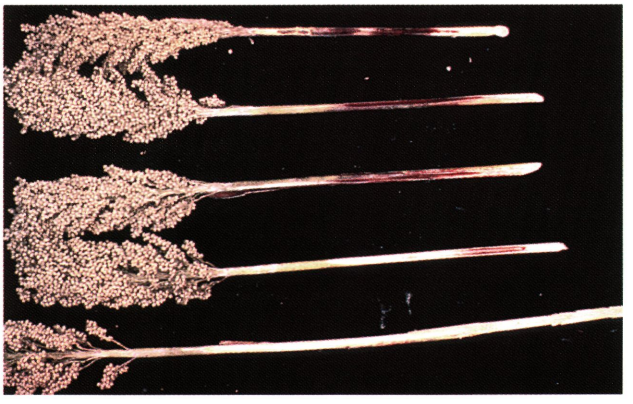

Photo 26. Drift of lipid synthesis inhibitors to susceptible grass crops such as grain sorghum, can partially damage and weaken the stem without completely destroying the tissue, as shown on these sorghum stems.

Photo 27. Cell membrane disruptors cause foliar burn type symptoms, as exhibited by this soybean leaf treated with Cobra herbicide.

Photo 28. Aim and Resource herbicides frequently cause foliar burn type symptoms on corn or sorghum, especially with hot, humid conditions following application.

Cell Membrane Disrupters

Protoporphyrinogen Oxidase (PPO) inhibitors (14):

Diphenylether	aciflourfen (Ultra Blazer) lactofen (Cobra) fomesafen (Reflex, Flexstar)
Aryl triazolinone	sulfentrazone (Authority, Spartan) carfentrazone (Aim)
Phenylphthalimide	flumiclorac (Resource)

Photosystem I Electron Diverters (22):

Bipyridilium	paraquat (Gramoxone Max) diquat (Reglone)

Photo 29. Paraquat drift will cause necrotic burn spots wherever the spray droplets come in contact with plant foliage, as with this wheat that was next to a treated field.

The cell membrane disrupters are primarily nontranslocated herbicides that are light activated. Paraquat and diquat are relatively nonselective chemicals used to control all existing vegetation and as preharvest desiccants. The PPO inhibitor herbicides provide selective broadleaf weed control in various crops. These herbicides quickly form highly reactive compounds in the plants that rupture cell membranes, causing the fluids to leak out. Thorough spray coverage is essential for good postemergence weed control. Because they are not translocated to the roots, these herbicides are ineffective for long-term perennial weed control.

Injury symptoms can occur within 1 to 2 hours after application, appearing first as water- soaked foliage, which is followed by browning (necrosis) of the tissue wherever the spray contacts the foliage (Photos 27 and 28). Symptoms will appear most quickly with bright, sunny conditions at application. Drift injury will appear as speckling on leaf tissue

Photo 30. Preemergence sulfentrazone treatments may cause a mottled chlorosis of foliage, especially with wet conditions on coarse-textured soils having high pH and low organic matter, as with these sunflowers treated with Spartan.

Photos 31 and 32. Pigment inhibitor herbicides cause a bleaching symptom when the chlorophyl in the leaves is destroyed (top). Command herbicide can carry over and damage small grains, especially in areas with higher application rates (bottom).

(Photo 29). Injury from soil applications or residues appear as a mottled chlorosis and necrosis (Photo 30).

Environmental and use considerations. The bipryidiliums are irreversibly adsorbed upon contact with the soil and have no soil activity. They are persistent in the soil, however, and could potentially move with the soil. The diphenylether herbicides and Resource are shorter-lived, and not as tightly bound to the soil.

Pigment Inhibitors

Diterpene inhibitors (13):

Isoxazolidinone	clomazone (Command)

Hydroxyphenylpyruvate dioxygenase (HPPD) synthesis inhibitors (28):

Isoxazole	isoxaflutole (Balance)

Phytoene desaturase (PDS) synthesis inhibitors (12):

Pyridazinone	norflurazone (Zorial)

The pigment inhibitor herbicides interfere with the production and protection of photosynthetic pigments. Ultimately, chlorophyl production is inhibited, and plant foliage turns white. Although injury symptoms are similar with these herbicides, the specific site of action is different. These herbicides are primarily xylem translocated, so they

Photo 33. Susceptible species to pigment inhibitors will have white or bleached foliage following emergence, as shown by this crabgrass emerging from soil treated with Balance herbicide.

Photo 34. Corn injury from Balance can result following cool, wet conditions on low organic matter, high pH soils.

are most effective as preemergence treatments. However, emerged plants also will turn white when exposed to a pigment inhibitor herbicide.

Injury symptoms from the pigment inhibitor herbicides are expressed as white to translucent color of the new foliage (Photos 31, 32, 33 and 34).

Environmental and use considerations. Drift to nontarget plants will cause foliage to turn white. Balance herbicide is quickly converted to a fairly soluble and persistent herbicidally active metabolite in the soil. Balance use is restricted on coarse textured soils with a shallow water table.

Herbicides, active ingredients, WSSA site of action classification, and labeled crops.

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Accent	nicosulfuron	2 ALS	corn
Accent Gold	rimsulfuron nicosulfuron flumetsulam	2 ALS 2 ALS 2 ALS	corn
Acquire	glyphosate	9 EPSP	fallow, Glyphosate-resistant soybeans
Aim	carfentrazone	14 PPO	corn, sorghum, wheat
Ally	metsulfuron	2 ALS	small grains, pasture, CRP
Amber	triasulfuron	2 ALS	small grains, pasture
Assure II	quizalofop	1 ACCase	soybeans, cotton
Atrazine	atrazine	5 PSII(A)	corn, sorghum, fallow
Authority	sulfentrazone	14 PPO	soybeans
Axiom	flufenacet metribuzin	15 SHT/RT 5 PS II(A)	corn, soybeans
Axiom AT	flufenacet metribuzin, atrazine	15 SHT/RT 5 PSII(A)	corn
Backdraft	imazaquin glyphosate	2 ALS 9 EPSP	Glyphosate-resistant soybeans
Balan	benefin	3 MT	alfalfa
Balance (PRO)	isoxaflutole	28 HPPD	corn
Banvel	dicamba	4 GR	corn, sorghum, small grains, fallow, pasture
Basagran	bentazon	6 PSII(C)	soybeans, corn, sorghum
Basis	rimsulfuron thifensulfuron	2 ALS 2 ALS	corn
Basis Gold	rimsulfuron nicosulfuron atrazine	2 ALS 2 ALS 5 PSII(A)	corn
Beacon	primisulfuron	2 ALS	corn
Bicep MAGNUM	metolachlor atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Bison	bromoxynil MCPA	6 PSII(C) 4 GR	small grains
Bladex	cyanazine	5 PSII(A)	corn

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Boundary	*S*-metolachlor metribuzin	15 SHT/RT 5 PSII(A)	soybeans
Broadstrike+Treflan	flumetsulam trifluralin	2 ALS 3 MT	soybeans
Bronate	bromoxynil MCPA	6 PSII(C) 4 GR	small grains
Buctril	bromoxynil	6 PSII(C)	corn, sorghum, small grains alfalfa
Buctril+Atrazine	bromoxynil atrazine	6 PSII(C) 5 PSII(A)	corn, sorghum
Bullet	alachlor atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Butyrac	2,4-DB	4 GR	soybeans, alfalfa
Canopy	chlorimuron metribuzin	2 ALS 5 PSII(A)	soybeans
Canopy XL	chlorimuron sulfentrazone	2 ALS 14 PPO	soybeans
Celebrity Plus	nicosulfuron dicamba, diflufenzopyr	2 ALS 4 GR	corn
Clarity	dicamba	4 GR	corn, sorghum, small grains
Classic	chlorimuron	2 ALS	soybeans
Cobra	lactofen	14 PPO	soybeans
Command	clomazone	13 DITERP	soybeans, fallow
Command Extra	clomazone sulfentrazone	13 DITERP 14 PPO	soybeans
Commence	clomazone trifluralin	13 DITERP 3 MT	soybeans
Cotoran	fluometuron	5 PSII(B)	cotton
Credit	glyphosate	9 EPSP	fallow, Glyphosate-resistant crops
Crossbow	triclopyr 2,4-D	4 GR 4 GR	pasture
Curtail	clopyralid 2,4-D	4 GR 4 GR	small grains, pasture
Define	flufenacet	15 SHT/RT	corn
Degree	acetochlor	15 SHT/RT	corn
Degree Xtra	acetochlor atrazine	15 SHT/RT 5 PSII(A)	corn

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Domain	flufenacet metribuzin	15 SHT/RT 5 PSII(A)	soybeans
Distinct	dicamba diflufenzopyr	4 GR 4 GR	corn
DoublePlay	acetochlor EPTC	15 SHT/RT 8 SHT	corn
Dual MAGNUM	*S*-metolachlor	15 SHT/RT	corn, sorghum, soybeans
Epic	flufenacet isoxaflutole	15 SHT/RT 28 HPPD	corn
Eptam	EPTC	8 SHT	alfalfa
Eradicane	EPTC	8 SHT	corn
Escort	metsulfuron	2 ALS	pasture, noncropland
Exceed	primisulfuron prosulfuron	2 ALS 2 ALS	corn
Express	tribenuron	2 ALS	small grains
Extreme	imazethapyr glyphosate	2 ALS 9 EPSP	Glyphosate-resistant soybeans
Fallow Master (BS)	glylphosate dicamba	9 EPSP 4 GR	fallow
FieldMaster	glyphosate acetochlor atrazine	9 EPSP 15 SHT/RT 5 PSII(A)	corn
Finesse	chlorsulfuron metsulfuron	2 ALS 2 ALS	small grains
FirstRate	cloransulam	2 ALS	soybeans
Flexstar	fomesafen	14 PPO	soybeans
Freedom	alachlor trifluralin	15 SHT/RT 3 MT	soybeans
Frontier	dimethenamid	15 SHT/RT	corn, sorghum, soybeans
FulTime	acetochlor atrazine	15 SHT/RT 5 PSII(A)	corn
Fusilade DX	fluaziflop	1 ACC	soybeans, cotton
Fusion	fluaziflop fenoxaprop	1 ACC 1 ACC	soybeans, cotton
Galaxy	acifluorfen bentazon	14 PPO 6 PSII(C)	soybeans
Garlon	triclopyr	4 GR	pasture

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Gauntlet	cloransulam sulfentrazone	2 ALS 14 PPO	soybeans
Glean	chlorsulfuron	2 ALS	small grains
Glyfos	glyphosate	9 EPSP	fallow, Glyphosate-resistant soybeans
Glyphomax	glyphosate	9 EPSP	fallow, Glyphosate-resistant soybeans
Gramoxone Max	paraquat	22 ED	fallow
Grazon P+D	picloram, 2,4-D	4 GR 4 GR	pasture
Guardsman	dimethenamid atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Harmony Extra	thifensulfuron tribenuron	2 ALS 2 ALS	small grains
Harness	acetochlor	15 SHT/RT	corn
Harness Xtra	acetochlor atrazine	15 SHT/RT 5 PSII(A)	corn
Hoelon	diclofop	1 ACC	small grains
Hornet	flumetsulam clopyralid	2 ALS 4 GR	corn
Hyvar	bromacil	5 PSII(A)	noncropland
Karmex	diuron	7 PSII(B)	alfalfa, cotton, noncropland
Laddok	bentazon atrazine	6 PSII(C) 5 PSII(A)	corn, sorghum
Landmaster BW	glyphosate 2,4-D	9 EPSP 4 GR	fallow
Lariat	alachlor atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Lasso	alachlor	15 SHT/RT	corn, sorghum, soybeans
Leadoff	dimethenamid atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Liberty	glufosinate	10 GS	Liberty resistant corn and soybeans
Liberty ATZ	glufosinate atrazine	10 GS 5 PSII(A)	Liberty resistant corn
Lightning	imazethapyr imazapyr	2 ALS 2 ALS	Clearfield (imi) corn

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Marksman	dicamba atrazine	4 GR 5 PSII(A)	corn, sorghum
Maverick Pro	sulfosulfuron	2 ALS	wheat
MCPA	MCPA	4 GR	small grains
Moxy	bromoxynil	6 PSII(C)	corn, sorghum, small grains, alfalfa
Moxy+Atrazine	bromoxynil atrazine	6 PSII(C) 5 PSII(A)	corn, sorghum
NorthStar	primisulfuron dicamba	2 ALS 4 GR	dicamba
OpTill	dimethenamid dicamba	15 SHT/RT 4 GR	corn
Oust	sulfometuron	2 ALS	noncropland
Outlook	*P*-dimethenamid	15 SHT/RT	corn, sorghum, soybeans
Paramount	quinclorac	4 GR	sorghum, fallow
Partner	alachlor	15 SHT/RT	corn, sorghum, soybeans
Peak	prosulfuron	2 ALS	sorghum, wheat
Permit	halosulfuron	2 ALS	corn, sorghum
Pinnacle	thifensulfuron	2 ALS	soybeans
Plateau	imazapic	2 ALS	noncropland
Poast, Poast Plus	sethoxydim	1 ACC	soybeans, alfalfa, cotton, sunflower
Pramitol	prometon	5 PSII(A)	noncropland
Princep	simazine	5 PSII(A)	noncropland, corn
Prowl	pendimethalin	3 MT	corn, soybeans, sunflower, cotton
Pursuit	imazethapyr	2 ALS	soybeans, corn, alfalfa
Pursuit Plus	imazethapyr pendimethalin	2 ALS 3 MT	soybeans
Python	flumetsulam	2 ALS	corn, soybeans
Ramrod	propachlor	15 SHT/RT	corn, sorghum
Ramrod/Atrazine	propachlor atrazine	15 SHT/RT 5 PSII(A)	corn, sorghum
Raptor	imazamox	2 ALS	soybeans
Rave	triasulfuron dicamba	2 ALS 4 GR	wheat

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Ready Master ATZ	glyphosate atrazine	9 EPSP 5 PSII(A)	corn
Reflex	fomesafen	14 PPO	soybeans
Reglone	diquat	22 ED	aquatic, crop dessicant
Remedy	triclopyr	4 GR	pasture
Resource	flumiclorac	14 PPO	corn, soybeans
Roundup Ultra	glyphosate	9 EPSP	fallow, Glyphosate-resistant crops
Sahara	imazapyr diuron	2 ALS 7 PSII(B)	noncropland
Scepter	imazaquin	2 ALS	soybeans
Select	clethodim	1 ACC	soybeans, alfalfa, cotton
Sencor	metribuzin	5 PSII(A)	soybeans, corn, small grains, alfalfa
Shotgun	atrazine 2,4-D	5 PSII(A) 4 GR	corn, sorghum
Sinbar	terbacil	5 PSII(A)	alfalfa
Sonalan	ethalfluralin	3 MT	soybeans, sunflower
Spartan	sulfentrazone	14 PPO	sunflower
Spike	tebuthiuron	7 PSII(B)	noncropland
Spirit	primisulfuron prosulfuron	2 ALS 2 ALS	corn
Squadron	pendimethalin imazaqin	3 MT 2 ALS	soybeans
Staple	pyrithiobac	2 ALS	cotton
Starane	fluroxypyr	4 GR	small grains
Starane+Salvo	fluroxypyr 2,4-D	4 GR 4 GR	wheat
Steadfast	nicosulfuron rimsulfuron	2 ALS 2 ALS	corn
Steel	pendimethalin imazethapyr imazaqin	3 MT 2 ALS 2 ALS	soybeans
Stinger	clorpyralid	4 GR	small grains, pasture, corn
Storm	acifluorfen bentazon	14 PPO 6 PSII(C)	soybeans
Surpass	acetochlor	15 SHT/RT	corn

Herbicide	Active Ingredients	WSSA Site of Action Classifications*	Labeled Crops
Sutan+	butylate	8 SHT	corn
Synchrony STS	chlorimuron thifensulfuron	2 ALS 2 ALS	STS soybeans
Topnotch	acetochlor atrazine	15 SHT/RT 5 PSII(A)	corn
Tordon	picloram	4 GR	pasture, noncropland, fallow
Touchdown	glyphosate	9 EPSP	fallow, Glyphosate-resistant crops
Tough	pyridate	6 PSII(C)	corn
Treflan	trifluralin	3 MT	soybeans, sunflower, alfalfa, cotton
Tri-Scept	trifluralin, imazaquin	3 MT 2 ALS	soybeans
Ultra Blazer	acifluorfen	14 PPO	soybeans
Velpar	hexazinone	5 PSII(A)	alfalfa, noncropland
Zorial	norflurazon	12 PDS	alfalfa, cotton
2,4-D	2,4-D	4 GR	corn, sorghum, small grains, pasture

WSSA Site of Action and Classification Number:

* 1 ACCase = acetyl-CoA carboxylase inhibitor
2 ALS= acetolactate synthase inhibitor
3 MT= microtubule assembly inhibitor
4 GR= growth regulator
5 PSII(A)= photosystem II, binding site A inhibitor
6 PSII(C)= photosystem II, binding site C inhibitor
7 PSII(B)= photosystem II, binding site B inhibitor
8 SHT= shoot inhibitor
9 EPSP= enolpyruvyl-shikimate-phosphate synthase inhibitor
10 GS= glutamine synthetase inhibitor
12 PDS= phytoene desaturase synthesis inhibitor
13 DITERP= diterpene inhibitor
14 PPO= protoporphyrinogen oxidase inhibitor
15 SHT/RT= shoot and root inhibitor
22 ED= photosytem 1 electron diverter
28 HPPD= hydroxyphenylpyruvate dioxygenase synthesis inhibitor

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