Why Herbicides Are Selective

David W. Cudney
Department of Botany and Plant Sciences
University of California, Riverside, CA 92521-0124

Selective herbicides have been used extensively since the introduction of 2,4-D in the late '40s. They have been one of the miracles of modem agriculture, releasing thousands of people from the drudgery of hand weeding. A selective herbicide is one that kills or retards the growth of an unwanted plant or "weed" while causing little or no injury to desirable species. 2,4-D used in turf will kill many of the broadleaf weeds that infest turf while not significantly injuring the turfgrass. But selectivity is a fickle, dynamic process. Excessive rates of 2,4-D applied to stressed turfgrass may injure the turf.

Selectivity has always depended on proper herbicide application. Normally herbicides work selectively within a given rate of application. Too little herbicide and no weed control, too much and crop injury may occur. But selectivity is more complex than this. It is a dynamic process that involves the interaction of the plant, the herbicide, and the environment.

The Plant

Factors that involve plant response include: genetic inheritance, age, growth rate, morphology, physiology, and biochemistry. The genetic make-up of a plant determines how that plant responds to herbicides and its environment. The age of the plant often determines how well an herbicide works, older plants are generally much more difficult to control than seedlings. Preemergence herbicides often work only on plants during the germination process and will have little effect on older plants. Plants which are growing rapidly are usually more susceptible to herbicides.

The morphology of a plant can help to determine its susceptibility to herbicides. Annual weeds in a deep-rooted crop can be controlled because the herbicide is concentrated in the first inch of soil where the weeds and weed seeds are. Weeds with exposed growing points may be killed by contact sprays, while grasses with protected growing points may be burned back, but escape permanent injury. Certain leaf properties can allow better spray retention and thus better kill (broadleaf species vs. grasses or hairy vs. smooth leaves). Sprays tend to be retained on pigweed and mustard leaves and bounce off of onion or grass species.

The physiology of a plant can determine how much of an herbicide will be absorbed onto the plant and the speed with which it is transported to its site of action. Plants with thick waxy cuticles or hairy leaf surfaces may not absorb sufficient herbicide to be injured. Wetting agents in herbicide formulations are used to combat these leaf characteristics and increase absorption. The transport rate of herbicides in plants varies. Usually susceptible plants transport herbicide more readily than resistant ones. Some plants can adsorb herbicides along the transport pathway, preventing them from reaching their site of action.

Biochemical reactions also account for selectivity. Most herbicides have a biochemical reaction within susceptible plants which accounts for their herbicidal activity. They may bind to critical enzymes within susceptible plants and block important metabolic processes (glyphosate), they may block photosynthesis (diuron) or respiration, or they may affect cell division (trifluralin). Herbicides may be absorbed as relatively innocuous chemicals (2,4-DB) and activated to deadly compounds (2,4-D) within susceptible plants. Other herbicides (atrazine) may be detoxified within some plants (com) while killing weeds which fail to metabolize the herbicide.

The Herbicide

Herbicides are quite specific in their structures as to whether or not herbicidal activity is possible. Slight changes in conformation or structure will alter herbicidal activity. Trifluralin and benefin differ in only a

methyl group moved from one side of the molecule to the other, yet trifluralin is about twice as active as benefin. Esters of phenoxy (MCPP etc.) acids are usually much more active than are amines. The manner of formulation of an herbicide can affect its selectivity. The most extreme case of this might be granular formulations which bounce off desirable plants to reach the soil where they then limit germinating weeds. Other substances known as adjuvants or surfactants are often added to improve the application properties of a liquid formulation and increase activity.

The manner in which an herbicide is applied can affect its selectivity. When a broad-spectrum postemergence herbicide like glyphosate is applied as a shielded, directed, or wicked application within a susceptible crop, susceptible foliage is avoided and selectivity is achieved with this normally non-selective herbicide.

Herbicides can be grouped into families based on the type of action that they have within affected plants (their mode of action). Herbicides which affect similar sites or processes within affected plants produce similar injury symptoms. The herbicide families listed below are grouped on the basis of how they affect plants:

1. The Growth Regulator Herbicides (2,4-D, MCPP, dicamba, and triclopyr). These are mostly foliar applied herbicides which are systemic and translocate in both the xylem and phloem of the plant. They mimic natural plant auxins, causing abnormal growth and disruption of the conductive tissues of the plant. The injury from this family of herbicides consists of twisted, malformed leaves and stems.
2. The inhibitors of amino acid synthesis (glyphosate, halosulfuron, imazethapyr, and sulfometuron). Both foliar and soil applied herbicides are in this family. Glyphosate translocates in the phloem with photosynthate produced in the leaves. Others in this family move readily after root or foliar absorption. These herbicides inhibit certain enzymes critical to the production of amino acids. Amino acids are the building blocks of proteins. Once protein production stops, growth stops. Symptoms are stunting and symptoms associated with lack of critical proteins.
3. Cell membrane disrupters - with soil activity (oxyfluorfen, lactofen, and acifluorfen). Soil and foliar applied with limited movement in soil. These herbicides enter the plant through leaves, stems, and roots, but are limited in their movement once they enter the plant. Membrane damage is due to lipid peroxidation. Symptoms are necrosis of leaves and stem.
4. Lipid biosynthesis inhibitors (diclofop, fluazifop, sethoxydim, and clethodim). Foliar applied Diclofop has both soil and foliar activity. Herbicides in this family move in both the xylem and phloem of the plant and inhibit enzymes critical in the production of lipids. Lipids are necessary to form plant membranes which are essential to growth and metabolic processes. Symptoms include stunting and death of tissue within the growing points of plants.
5. Pigment inhibitors (norflurazon, fluridone, and amitrol). Soil applied and move in the xylem except amitrol, which moves in both phloem and xylem. These herbicides inhibit carotinoid biosyntehsis, leaving chlorophyll unprotected from photooxidation. This results in foliage which lacks color. Symptoms include albino or bleached appearance of foliage.
6. Growth inhibitors of shoots (thiocarbamate herbicides including: EPTC, cycloate, pebulate, and molinate). Soil applied and somewhat volatile, requiring incorporation. Enter the plant through the roots and translocated through the xylem with the transpiration stream to the growing points in the shoot. Mode of action is unclear, but affects developing leaves in growing points of susceptible plants. Symptoms include stunting and distortion of seedling leaves.
7. Herbicides which disrupt cell division (trifluralin, DCPA, dithiopyr, oryzalin, pronamide, pendimethalin, and napropamide). All are soil applied, with limited movement in the soil. Absorbed through roots or emerging shoot tips. Once absorption takes place, movement is limited (site of action is near the site of absorption). These herbicides inhibit cell division or mitosis, except pronamide and napropamide which stop cell division before mitosis. Symptoms include stunting and swollen root tips.
8. Cell membrane disrupters - no soil activity (paraquat, diquat, glufosinate, acids, oils, soaps). These herbicides are foliar applied with no soil activity. They enter the plant through the leaves and stems and do not move significantly within the plant once absorbed. These herbicides either act directly on cell

membranes (acids, soaps. oils) or react with a plant process to form destructive compounds which result in membrane damage. Symptoms include rapid necrosis of the leaves and stem.

9. Inhibitors of photosynthesis (atrazine, simazine, metribuzin, cyanazine, prometryn, diuron, linuron, tebuthiuron, and bromacil). These are soil applied herbicides, however, all except simazine also have foliar activity. They move readily in the plant in the xylem with the transpiration stream where they concentrate in the leaves at the site of photosynthesis. Once there they block the electron transport system of photosynthesis, causing a build up of destructive high energy products which destroy chlorophyll and ultimately the leaf tissues. Symptoms include chlorotic (yellowed) leaves which become necrotic.

The Environment

There are many ways that the environment interacts with herbicide selectivity. The soil determines how much of soil applied herbicides are available for activity. Sandy soils, with low organic content, are much more active and conversely less selective than clay soils with high organic content at a given rate of herbicide application. Irrigation or rainfall amount and timing influence the depth to which herbicides may move in the soil and plant growth and stress, all of which can increase or decrease herbicide selectivity. Temperature affects the rate of herbicide transport, the rate of biochemical reactions, plant growth, plant stress, and ultimately herbicide selectivity. Wind, relative humidity, insects, plant pathogens, and nutritional status also affect plant growth and stress which can increase or decrease herbicide selectivity.

Herbicide selectivity is truly a dynamic process. It involves complex interactions between the plant, the herbicide and the environment.

Suggested Reading

Ashton, Floyd M. and W. A. Harvey. 1976. Selective chemical weed control. University of California bulletin 1919. 17 pages.
Warren, G. F., F D. Hess, et al. 1995 Herbicide action short course at Purdue. Syllabus. 787 pages.
Tickes, Barry, D. W. Cudney, and C. L. Elmore. 1996. Herbicide injury symptoms. University of Arizona Bulletin 195021.