How Herbicides Work: Part I - Chemical Classification

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INTRODUCTION

Weeds are the top pest problem in home lawns and are the second most important pest problem on golf turf in Wisconsin (WASS and UWEX, 2001). Though good cultural management can effectively reduce most weed populations, the traffic, disease, and insect injuries on golf turf provide opportunities for weed encroachment in even the most highly managed golf turf. Herbicides are the last line of defense against weeds, but aren't consistently effective. This article is the first of a series on understanding herbicide activity for better control: the current article focuses on the chemical classifications of herbicide and describes the basis for selective weed control and herbicide resistance. Future articles in the series will discuss factors that influence herbicide efficacy, fate, and environmental impact.

WHAT'S IN A NAME?

Herbicides, like other pesticides, have three types of names. The **chemical** name is assembled by chemists based on the structure of the compound. The chemical name is developed following rules of nomenclature (naming) established by scientific organizations such as the International Union of Pure and Applied Chemistry (IUPAC, 2003). Certain chemical structures, ranging from chains of atoms to cyclic structures, have been assigned permanent names. Nomenclature rules define the position of specific groups of molecules or atoms on a large molecule by assigning them a numbered position. Since chemical names are long and complex, companies that produce chemicals for pesticide use must propose a **common name** for the chemical to the Environmental Protection Agency (EPA) as part of the registration process. To entice buyers, and because a given active ingredient may be sold in different concentrations or formulations, a herbicide product will be given a **trade** name by the distributor (seller). In many cases the distributor is someone other than the manufacturer of the chemical.

An example of chemical, common, and trade names can be described using 2,4-D. The chemical name is 2,4-dichlorophenoxyacetic acid (Fig. 1). The central structure is a ring of six carbon atoms which is shown as a hexagon (the circle inside the hexagon indicates there are two bonds between each carbon atom). This is the most stable portion of 2,4-D. The ring structure has an oxygen atom (O) attached to it, making the central molecule a phenoxy group. The oxygen atom has an acetic acid (CH2-COOH) molecule attached to it, modifying the base name to a phenoxyacetic acid. (Incidentally, acetic acid is the chemical which gives vinegar its acidic flavor. At high enough concentrations, acetic acid itself can be an effective contact herbicide. See *The Grass Roots* 31(6):5-7, "Hard cider? Kill some weeds with it!) The carbon with the oxygen attached to it is assigned the number 1 position in the ring. Chlorine (Cl) atoms are attached to carbon atoms number 2 and 4. Thus, the **chemical** name 2,4-dichlorophenoxyacetic acid. The **common** name, of course, is 2,4-D. The chemical is used in many herbicide formulations under **trade** names such as Weedone LV-4, Trimec, etc. Trade names change routinely. Common names almost never change. Chemical names never change.

UNDERSTANDING HERBICIDE ACTIVITY

Herbicides can affect one or more plant parts or enzymes. The plant part or enzyme(s) affected is/are known as the **site of action**. The **mode of action** is the physiological basis for herbicide activity, e.g., a herbicide that affects photosynthesis has a mode of action known as a photosynthetic inhibitor, even though its site of action may be a particular enzyme necessary for photosynthesis. Both the site of action and mode of action help determine the effectiveness, length of time, and other variables associated with herbicide application and the impact of cultural management practices. For example, rainfall or removal of leaf

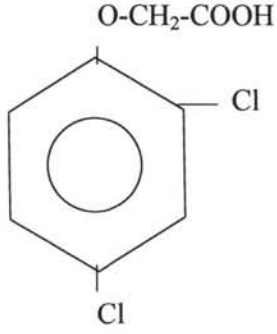

Figure 1. Chemical structure of 2,4-dichlorophenoxyacetic acid (2,4-D).

tissue immediately after application of a slow-acting herbicide may remove a critical amount of the herbicide before it can act to kill the growing point of the weed.

The mode of action is important because many herbicides have similar modes of action yet may be in different chemical classes. The EPA, under the Food Quality Protection Act, is currently reviewing all pesticides and grouping pesticides within a similar chemical class when deciding to allow (re)registration of individual chemicals. Routine use of herbicides that have similar sites of action, especially if they target the same enzyme, can lead to herbicide resistant weeds or enhanced microbial degradation.

Herbicides are placed in a specific chemical class based on their chemical structure. Understanding resistance mechanisms is difficult, though, because herbicides within a chemical class may act differently. For example, bensulide and glyphosate are both organophosphates. Bensulide inhibits cell division (meristem inhibitor) and is used as a pre-emergent herbicide for control of annual weeds while glyphosate inhibits an enzyme needed for amino acid production and is used as a post-emergent. Specific characteristics of each chemical class are outlined in Table 1.

TYPES OF HERBICIDAL ACTIVITY

Herbicides may have one or more modes of action (Callahan, 1994; Penner, 1994). The most common types are described below. In some cases, the mode of action is restricted to a specific site of the plant.

Auxin agonists. Most of the herbicides used for broadleaf weed control in turfgrass are auxin agonists, including those in the phenoxy and pyridine chemical classes. Auxin agonists mimic and/or enhance production of naturally-occuring plant hormones. The chemicals cause uncontrolled growth, resulting in twisting and curling of weed stems and leaves. Weed death is slow because the plant basically grows itself to death. Auxin agonists are the mainstay of the home lawn weed control industry but are also important for golf courses. While some phenoxys like 2,4-D can be phytotoxic to creeping bentgrass, chemicals in the pyridine class have less toxicity and in some cases have been developed as formulations specifically for broadleaf weed control in bentgrass fairways (e.g., Lontrel).

Photosynthetic inhibitors. Some of the most potent and rapidly-acting herbicides disrupt photosynthesis. Without the energy produced by photosynthesis, plants will eventually die. Benzothiadiazoles inhibit the Photosystem II (PS II) portion of

Table 1. Herbicides commonly used in cool season turf management.

				Examples	
Class	**Primary Mode (site) of action**	**Weeds controlled**	**Timing**	**Common name**	**Trade name**
Arsenicals	Photosynthesis inhibitor	Annual grasses	Post-emergent	Monosodium methanearsonate (MSMA)	MSMA
Aryloxyphenoxy propionates	Fatty acid synthesis (ACCase inhibitor)	1) Grasses 2) Annual grasses	Post-emergent	1) Fluazifop-butyl 2) Fenoxaprop	1) Fusilade 2) Acclaim
Benzamides	Mitosis inhibitor (roots)	Annual bluegrass	Pre- and post-emergent	Pronamide	Kerb
Benzoic acids	Auxin agonist, DNA inhibitor, ethylene production	Broadleaves	Post-emergent	Dicamba*	Multiple trade names and combinations
Benzofuran	Lipid synthesis (cuticle inhibition)	Annual bluegrass	Post-emergent	Ethofumesate	Prograss
Benzothia-diazoles	Photosynthetic inhibitor (PS II)	Yellow nutsedge, broadleaves	Post-emergent	Bentazon	Basagran
Bypyridiliums	Photosynthetic inhibitor (PS I)	Non-selective	Post-emergent	Diquat	
Dinitroanilines	Mitotic inhibitor	Annual grasses & broadleaves	Pre-emergent	Benefin* Oryzalin* Trifluralin* Prodiamine Pendimethalin	*Multiple trade names and combinations Barricade Pre-M
Organophos-phates	1)Amino acid inhibitor (EPSP synthetase), 2) mitotic inhibitor	1) Non-selective or 2) Annual grasses & broadleaves	1) Post-emergent or 2) pre-emergent	1) Glyphosate 2) Bensulide	1) Roundup 2) Betasan
Oxadiazole	Chlorophyll inhibition	Annual grasses	Pre-emergent	Oxadiazon	Ronstar
Phenoxys	Auxin agonists	Broadleaves	Post-emergent	2,4-D* 2,4-DP* MCPA* MCPP*	Multiple trade names and combinations
Pyridines	Unknown, likely auxin agonist	Broadleaves	Post-emergent	Triclopyr* Clopyralid*	Confront Lontrel
Substituted ureas	Mitotic inhibitor (roots)	Annual grasses	Pre-emergent	Siduron	Tupersan
Sulfonyl ureas	Mitotic inhibitor	1) Tall fescue, perennial ryegrass 2) Yellow nutsedge	Post-emergent	1) Chlorsulfuron 2) Halosulfuron	1) Cavalier 2) Manage
	Miscellaneous compounds				
	Auxin agonist and enhanced ethylene production?	Annual grasses and broadleaves	Pre- and post-emergent	Quinclorac	Drive
	Fumigant Mitotic inhibitor	Non-selective Annual grasses	Pre-plant Pre- and partial post-emergent	Dazomet Dithiopyr	Basamid Dimension

Adapted from Penner, 1994.

*Usually sold in mixtures with other active ingredients.

Note: Many of the pre-emergent compounds inhibit germination of multiple species, including broadleaf weeds, though their primary use in cool-season turf is for annual grasses.

photosynthesis by binding to the D1 protein and reducing electron transport. Bypyridiliums act on another stage of photosynthesis, PS I, and kill weeds quickly because their activity generates highly energized "free radicals" which literally bounce around a cell and destroy membranes, rapidly and completely eliminating all types of metabolic activity. Many photosynthetic inhibitors turn sensitive plants white.

Amino acid inhibitors. Amino acids are the building blocks of proteins (many of which are enzymes) which are the vehicle for production of all other plant compounds, cell structures, and organs. Glyphosate, for example, interrupts the production of phenylalanine, tyrosine, and tryptophan by blocking the activity of the EPSP synthase enzyme. Glyphosate-resistance has been developed in some transgenic plants, including creeping bentgrass, by inserting a different form of the EPSP enzyme (from bacteria) which is not affected by glyphosate.

Fatty acid inhibitors. Chemicals in this class inhibit the activity of a specific enzyme known as ACCase. Chemicals that inhibit only one specific enzyme in a target organism often allow the development of pesticide resistance when the chemical is not properly rotated with chemical of different activity. Although some agronomic weeds have developed resistance to ACCase inhibitors, this has not been common in turf in part because herbicides are used less frequently or are rotated; in addition, those labeled for turf may also have secondary modes of action such as membrane disruption, auxin inhibition, or meristematic inhibition.

Lipid synthesis inhibitors. Herbicides such as ethofumesate (Prograss) destroy weeds by preventing production of lipids which have various functions in the plant, including the development of the waxy cuticle on leaf surfaces. Plants without a proper cuticle are more likely to dry out and die, or be attacked by pathogenic fungi, than plants with a fully formed cuticle.

Mitotic inhibitors. Mitosis is the process of cell division followed by formation of new cells, a form of asexual reproduction. Mitosis is absolutely necessary for plant growth. Many pre-emergent herbicides stop mitosis in the meristem (region of active mitosis) of new roots during seedling germination, effectively stopping root growth. Without roots, the germinating seedlings die without usually ever emerging from the soil. Many mitotic inhibitors are actually contact herbicides. Sulfonyl ureas can also prevent cell division but these herbicides are applied post-emergent and

are usually selective for certain grasses and broadleaves.

Ethylene production. Some herbicides enhance ethylene production in addition to having other modes of action. Ethylene is a plant-produced hormone and a certain amount is necessary for normal growth and development. Herbicides that enhance ethylene production, though, cause uncontrolled effects from it such as rapid cell death and reduced plant growth. Quinclorac (Drive) is a recently labeled compound for turf which has both ethylene-stimulatory and auxin-mimicry properties (Hawes, 1999). It is especially effective for post-emergent control of crabgrass.

CONCLUSION

There are additional types of herbicide activities and many of those described above may be much more complex than indicated. However, the above descriptions should serve as a guide to understanding herbicide activity. While herbicide resistance has been reported for many agronomic weeds, herbicide resistance in turf is currently of minimal importance. Part of the reason may be underreporting of non-control; lack of resistance may also be due to lower herbicide use (less consistent) and/or rotation of chemicals. Though herbicide resistance could in the future become more important in turf management, much more problematic is the loss of conventional herbicides due to the Food Quality Protection Act.

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