SERIES: How to Get the Most Out of Your Weed Management Program in Christmas Tree Production?

TOPIC: Understanding Herbicide Modes of Action

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Introduction:

For a successful Christmas tree weed management program, depending solely on non-chemical weed management methods may not be very effective. In Christmas tree production, growers generally depend on chemical weed control methods along with mechanical mowing. Herbicides play an important role and it is very crucial to understand how these chemicals work to choose the right product. The mode-of-action is the overall way herbicide affects a plant at the tissue or cellular level. It usually refers to the biological process or enzyme in which an herbicide acts to limit the growth of a specific plant (1). Although Mode of Action (MOA) is often used interchangeably with Site of Action (SOA), there is a difference between them. MOA is the overall way in which an herbicide kills a plant, whereas SOA is the biochemical or biophysical process in the plant that the herbicide disrupts to interfere with plant growth (2).

TABLE 1: *Various modes and sites of action as per Weed Science Society of America.[Adapted from source (2)].*

Mode of Action	WSSA Group	Site of Action
Lipid Synthesis Inhibitors	1	ACCase Inhibitors (acetyl-CoA carboxylase)
Amino Acid Synthesis Inhibitors	2	ALS Inhibitors (acetolactate synthase)
	9	EPSP Synthase Inhibitor (5-enolpyruvyl-shikimate-3-phosphate)
Growth Regulators (Synthetic auxins)	4	Various sites
	19	Auxin Transport
Photosynthesis Inhibitors	5	Photosystem II Inhibitors (serine 264 binders)
	6	Photosystem II Inhibitors (histidine 215 binders)
Nitrogen Metabolism	10	Glutamine Synthesis Inhibitor
Pigment Inhibitors	12	PDS Inhibitor (phytoene desaturase synthesis)
	13	DOXP Synthesis Inhibitors (1-deoxy-D-xyulose 5-phosphate)
	27	HPPD Inhibitors (hydroxyphenylpyruvate dioxygenase)
Cell Membrane Disrupters	14	PPO Inhibitors (protoporphyrinogen oxidase)
	22	Photosystem I Electron Diverters
Seedling Root Growth Inhibitors	3	Microtubule Inhibitors
Seedling Shoot Growth Inhibitors	15	Very Long-chain Fatty Acid Inhibitors

The Weed Science Society of America (WSSA) has developed a numerical classification system of herbicides based on their mode of action (Table 1). This classification is a fluid system, i.e., updates are made to the groups when new information and facts about the herbicides are discovered (2). It is essential to know about the site of action of herbicides for proper selection and rotation of herbicides, and also to lower the risk of developing herbicide-resistant weeds.

Some important WSSA herbicide groups are discussed further

Group 1 (ACCase inhibitors):

Acetyl-CoA carboxylase catalyzes the ATP-dependent formation of malonyl-CoA from acetyl-CoA and bicarbonate. This reaction is the first committed step in the biosynthesis of fatty acids, providing the essential substrate for fatty acid synthetase (3,4). This inhibition of fatty acid synthesis presumably blocks the production of phospholipids used in building new membranes required for cell growth. Fatty acids are essential components of plants' cell membranes. Failure to produce fatty acids arrests the production of new cells (2). Group 1 herbicides are generally used in grasses since they have little or no activity on broadleaves. Some commonly used active ingredients of group 1 are clethodim, cycloxydim, sethoxydim, and others.

Group 2 (Acetolactate Synthase inhibitors):

Group 2 herbicides in the WSSA classification inhibit the action of acetolactate synthase (ALS), an enzyme that facilitates the biosynthesis of the branched chain amino acids isoleucine, leucine, and valine. Amino acids are essential building blocks to proteins and are required for plant metabolism to function properly. ALS-inhibiting herbicides are readily absorbed by both the xylem and phloem to the site for action at the growing points. Selectivity is based on differential metabolism and/or an altered site of action.

Group 3 (Inhibitors of microtubule assembly):

Group 3 herbicides bind to tubulin; this herbicide-tubulin complex inhibits polymerization of microtubules at the assembly end of the protein-based microtubule but does not affect depolymerization of the tubule on the other end, leading to a loss of microtubule structure and function (5). These herbicides are absorbed by both roots and shoots of emerging seedlings, but these herbicides are not readily translocated. These herbicides are mitotic disrupters that inhibit cell division in plants. Some commonly used Group 3 herbicides are pendimethalin, oryzalin, dithiopyr, prodiamine, and others.

Group 4 (Synthetic Auxins):

These herbicides are similar to endogenous auxin (IAA). The primary action of these compounds is thought to be affecting the cell wall plasticity and nucleic acid metabolism by acidifying the cell wall by stimulating the activity of a membrane-bound ATPase proton pump. In low concentration, these herbicides stimulate RNA polymerase, increasing RNA, DNA, and protein biosynthesis. As a result, the cell division is uncontrolled, subsequently leading to vascular tissue destruction. In higher concentrations, these herbicides inhibit cell division and growth of meristematic tissues that accumulate photosynthate assimilates and herbicides from the phloem. These herbicides can also stimulate ethylene evolution, leading to epinasty in plants. Some common group 4 herbicides are 2,4-D, clopyralid, triclopyr, dicamba, MCPA, etc.

Group 5, 6, and 7 (Photosynthetic inhibitors at Photosystem II):

These herbicides interfere with the photosynthetic activities of the plant by binding the Qb-binding niche on the D1 protein of the photosystem II complex in chloroplast thylakoid membranes. The binding thus formed blocks electron transport and interrupts CO2 fixation and production of ATP and NADPH2, which are required for plant growth. Both Group 5 and 6 herbicides inhibit photosynthesis in plants. These herbicides are absorbed by shoots and roots but are translocated only in the xylem. The action of these herbicides only begins when they are exposed to light, and photosynthesis begins. Plant death is not due to starvation but due to the buildup of highly reactive molecules that destroy cell membranes (2). Some common Group 5 herbicides are simazine and atrazine etc. Bromoxynil and bentazon are commonly used Group 6 herbicides. The commonly used group 7 herbicides are diuron and propanil.

Group 9 [Inhibitors of 5-enolypyruvyl-shikimate-3-phosphate synthase (EPSP)]:

Group 9 herbicides inhibit the shikimic acid pathway. The inhibition of EPSP causes depletion of the aromatic amino acids: tryptophan, tyrosine, and phenylalanine, which are needed for protein synthesis. A very good example is glyphosate (6), which is readily absorbed through plant foliage and translocated in the phloem. It is a nonselective herbicide.

Group 12 (Inhibitors of Phytoene Desaturase):

Group 12 herbicides inhibit the action of Phytoene Desaturase (PDS) in carotenoid biosynthesis pathways (7,8). In a normal photosynthetic reaction, a singlet is produced, which is neutralized by Carotenoids and other molecules, but when the plant is treated with an herbicide like Fluridone lacks carotenoids and the singlet interferes with the chlorophyll, triggering a chain reaction of lipid damage. Eventually, the membranes and tissues are damaged. They are usually combined with other herbicide groups for better control of weeds.

Group 14 (Inhibitors of Protoporphyrinogen Oxidase):

These herbicides inhibit protoporphyrinogen oxidase, which is an enzyme required in heme biosynthesis. This inhibition of PPO causes the accumulation of PPIX, the first light-absorbing chlorophyll precursor. Light absorption by PPIX produces triplet state PPIX, which interacts with ground oxygen to form singlet oxygen. Both triplet PPIX and singlet oxygen can abstract hydrogen from unsaturated lipids, producing a lipid radical and initiating a chain reaction of lipid peroxidation. This results in loss of chlorophyll and carotenoids, leaky membranes, and the plant dies quickly (9). Lactofen, flumioxazin, oxyfluorfen , diquat, and paraquat, etc. are Group 14 herbicides being used commonly (6).

Group 15 [Very Long-Chain Fatty Acid (VLCFA) inhibitors]:

Group 15 herbicides are used as preemergence herbicides. They affect susceptible weeds before emergence, but do not inhibit seed germination or control emerged plants. A very good example of group 15 herbicide is dimethenamid-P which is commonly used for grasses and some broadleaf and for yellow nutsedge (2,6).

Group 19 (Auxin transport inhibitors):

These herbicides inhibit the transport of naturally occurring auxin, which causes indoleacetic acid accumulation in plants, disrupting the auxin balance needed for plant growth. Plants, when applied with Group 19 herbicides, exhibit rapid and severe hormonal effects such as epinasty. They are absorbed by plants through both roots and leaves and translocated by xylem and phloem. Naptalam and diflufezonpyr are group 19 herbicides that are used in the control of weeds.

Group 22 (Photosystem I inhibitors):

Diquat and paraquat fall under group 22 of WSSA classification. These herbicides accept electrons from photosystem I and get reduced to herbicide radical, which further reduces molecular oxygen to superoxide radicals. These radicals undergo a series of reactions to eventually form hydroxyl radical, which is highly reactive and destroys unsaturated lipids such as membrane fatty acids and chlorophyll. Hydroxyl radicals produce lipid radicals, which initiate a chain reaction of lipid oxidation leading to desiccation and wilting of plants.

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