Mode of Action of Herbicides

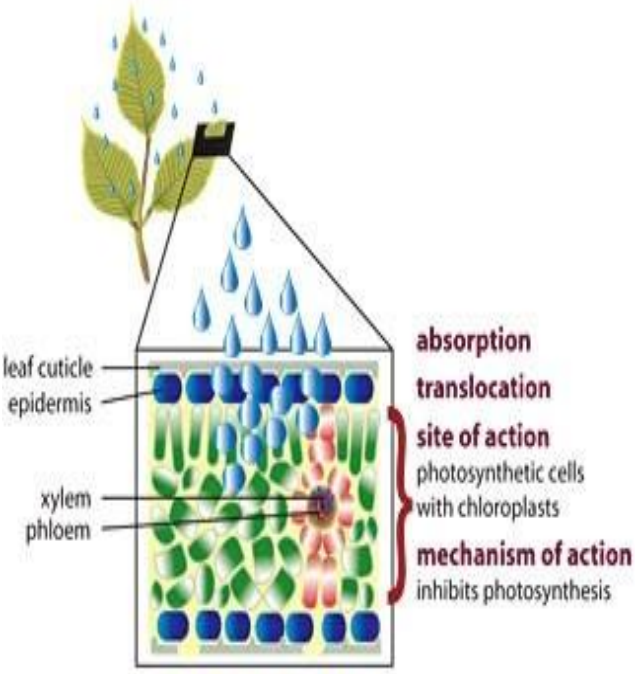
Herbicides kill or suppress plants by interfering with essential plant processes such as photosynthesis. All of the interactions between an herbicide and a plant from application to the final effect are referred to as the *mode of action*. Understanding the mode of action of an herbicide is essential in selecting the proper herbicide, diagnosing herbicide injury symptoms, preventing herbicide resistance problems, and avoiding non-target environmental impacts.

The mode of action involves

1. **Contact and absorption: -** Herbicides must contact the plant surface to be effective. Herbicides with limited mobility that are effective at the site where they contact the plant are known as ***contact herbicides***. Herbicides that must be absorbed and translocated to the site of action to be effective are called ***systemic herbicides***. Contact herbicides typically affect only the portion of the plant with which they come into physical contact. Contact herbicides are fast acting, and injury symptoms can appear within hours of application. Conversely, injury symptoms from systemic herbicides can take from several days to weeks to appear, but the entire plant may eventually be killed. ***Soil-applied*** herbicides are applied to the top few inches of the soil and eventually absorbed through root tissue, whereas ***foliar-applied*** herbicides are applied to leaves or stems. Most contact herbicides are foliar-applied, whereas systemic herbicides can be either soil- or foliar-applied. Choosing the appropriate herbicide depends upon target species biology, herbicide selectivity, application method, and site conditions. It is important to understand these factors to ensure that an effective herbicide is selected. For example, contact herbicides are most effective against annual invasive plants and in situations in which plant regrowth is not a concern. Conversely, systemic herbicides are more effective on perennial invasive plants and can limit regeneration of treated plants. Soil-applied herbicides are most effective on seedlings or germinating plants prior to their emergence above the

soil. Established plants may require a foliar-applied herbicide for effective control. Mature plant tissues absorb herbicides less easily than young plant tissues due to thickening of the outer tissues in older plants.

2. **Translocation:-** movement of the herbicide to the site of action. Systemic herbicides move, or translocate, from the point of application to the site of action through either the phloem (tissue that transports sugars from the leaves to the roots), xylem (tissue that transports water from the roots to the leaves), or through both. Some herbicides move more easily and farther within plants than others.
3. **Site of action:-** Specific location within the plant where the herbicide exerts toxicity at the cellular level. To be effective, an herbicide must reach the site of action. An herbicide binds to a specific location within the plant, typically a single protein, and as a result disrupts a physiological process essential for normal plant growth and development.



4. **Mechanism of action:-** specific biochemical or biophysical process that is affected by the herbicide.

The terms *mode of action* and *mechanism of action* are often used interchangeably. However, mechanism of action refers to the plant's specific biological process that is interrupted by the herbicide, whereas mode of action is a general term referring to all of the plant-herbicide interactions.

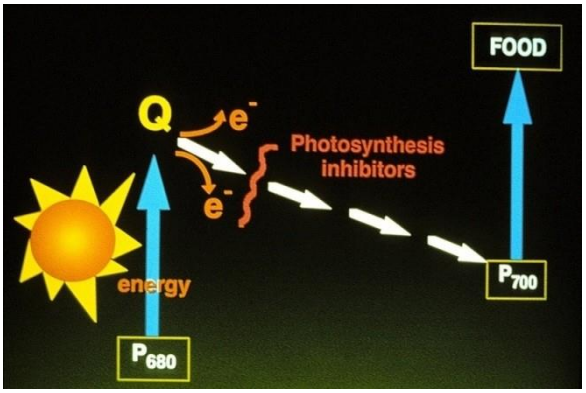
MECHANISM OF ACTION CLASSIFICATION:-

There are often many diverse chemical families within the same grouping, and to discuss each group on the basis of its chemistry would be confusing and in many cases redundant. Although we have a tremendous amount of specific information about most herbicides and how they result in plant death, it should be recognized that the exact mechanism of action of some herbicides is not known. Therefore, the classification

used is based on current knowledge related to the primary type or mechanism of action for each chemical and grouping.

Classification of Herbicides by Primary Type of Mechanism of Action

1) Photosynthesis Inhibitors



This group includes herbicides that are applied only to the soil, some that are applied only post-emergence to plant foliage, and some that can be applied both as soil and as foliar treatments. These herbicides inhibit electron transport in photosystem II of the photosynthetic reaction in plants, resulting in the formation of free radicals (potent biological oxidants) that attack and destroy the integrity of cell membranes. When soil applied, weed seeds germinate, their roots absorb the herbicide and translocate it in the xylem to the leaves, and the plant slowly dies as photosynthesis is inhibited. When applied post-emergence, the action is contact, requiring complete wetting of the foliage for complete kill. Susceptible plants turn yellow, then die from the bottom to the top. Leaves yellow between the veins and then turn brown from the base and outer leaf edges toward the center, eventually falling off the plant and leaving only a stem with an apical bud.

2) Pigment Inhibitors (No Chemical Family Recognized)

Pigment inhibitors are mostly applied as preplant or preemegence treatments, with the exception of amitrole (foliar) and fluridone in aquatics. These herbicides inhibit different enzymes in the carotenoid pigment biosynthetic pathway in the plant. Carotenoid pigments are important accessory plant pigments that protect chlorophyll from photooxidation. When carotenoids

are absent, chlorophyll is destroyed in the light and plants slowly die. Injury caused by these herbicides is a bleached white to translucent appearance of the leaves. Sometimes the bleaching is not complete on the entire leaf but will be interveinal with pink or red highlights along the margins. The most obviuos symptoms for pigment inhibitors herbicides is the suspectible plant tissue will turn white or very light green in color; new and older leaves may be affected. The most important comment is that carryover to susceptible crops is possible where spray overlap or misapplication has occurred. Volatility injury is also possible if the herbicide is not immediately incorporated into the soil after application.

3) Cell Membrane Disruptors and Inhibitors

a. Direct Effect on Membranes

b. Induce Lipid Peroxidation

   1. Photosynthesis involved

   2. Photosynthesis not involved

c. Inhibition of Glutamine Synthetase Glufosinate

There is a diversity of chemistry in this group of compounds, with most being applied as postemergence contact herbicides; however, oxidiazon, sulfentrazone, and oxyfluorfen have important uses as soil-applied preemergence herbicides. Although the specific site of inhibition in the plant varies among the cell membrane disruptor groups—group B1 (photosystem I), group B 2 [inhibition of protoporphyrinogen oxidase (PROTOX)], and group C (glutamine synthase)—plant death from these herbicides is rapid when they are applied to foliage. Complete coverage of the leaf is important for best activity, and the rate of plant death is more rapid under high light and warm environmental conditions. Injury symptoms include an initial appearance of water soaked tissue, followed by desiccation of leaf tissue caused by a disruption of cell membranes. Membranes are degraded by free radicals (potent biological oxidants) that form within plants as a result of the action of these herbicides.

4. Cell Growth Disruptors and Inhibitors

A. Mitotic Disruptors:

B. Inhibitors of shoots of emerging seedling: -

C. Inhibitors of Roots only of seedling: -

D. Inhibitors of Roots and Shoots of seedling: -

Herbicides in this classification inhibit root and/or shoot growth of emerging seedlings and are applied to the soil either as preemergence or preplant incorporated treatments. The mitotic disrupters inhibit the early steps in plant cell division responsible for chromosome separation and cell wall formation in plants, the root/shoot inhibitors deplete long-chain fatty acids from the plasma membranes of plants, whereas the specific mechanism shoot inhibitors is not known. *Mitoic disrupters* inhibit shoot elongation when effective, and susceptible weeds never see the light of day. Root inhibition is observed as root pruning, and roots can be swollen and expanded at the tip (club-shaped). The underground portion of the stem can be thickened and shortened, and stems often have callus growth thickenings at the soil surface and become brittle.

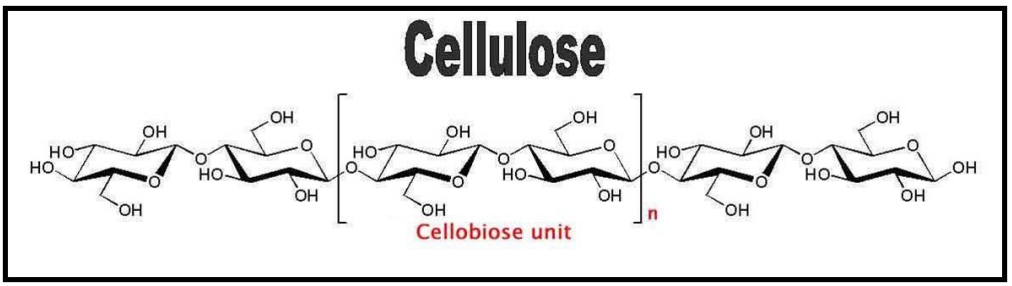
Swollen Hypocotyl

Inhibitors of roots only (C in this listing) or roots and shoots of seedlings (D) result in root pruning and growth inhibition but no root swelling. The inhibition of shoots by the carbamothioates (B) and the chloroacetamides and oxyacetamides (D) results in lack of seedling shoot emergence. If shoots do emerge, they tend to be twisted and leaves are tightly rolled, with stems sometimes rupturing and new growth protruding from the ruptured tissue.

(Leaves Failing to unfurl)

5. Cellulose Biosynthesis Inhibitors:-

The cellulose biosynthesis inhibitor herbicides are a diverse group of chemically unrelated compounds. The common herbicidal effect is either a direct or indirect inhibition of cellulose biosynthesis, which in effect leads to a lack of cell structure integrity. In most cases these herbicides are used for pre-emergence control and result in the inability of weed seedlings to grow. Symptoms include stunted growth and root swelling. Dichlobenil and isoxaben are used pre-emergence and are most effective against dicots, and quinclorac is used both pre-emergence and post-emergence. Quinclorac as a cellulose biosynthesis inhibitor is most active against monocots.



Cellulose is a simple, unbranched, linear polymer of glucose, arranged as a β-1,4-glucan, It is an integral part of the plant cell wall and hence cellulose synthesis is essential in order that cell wall can be synthesized. The cell wall determines to some extent both the morphology and the function of a cell, but even more importantly it controls the degree to which a cell can expand. The theory is that if cellulose biosynthesis is inhibited, then weakened cell walls will result, causing expansion of the cell and disruption of cellular processes. This in turn leads to abnormal or restricted growth and subsequent plant death. It appears that CBIs do not have a common site of action in the synthesis of cellulose, which is a process carried out by multi-enzyme complexes situated in the cell plasma membrane.

6. Growth Regulators:-

Growth regulator herbicides can be absorbed from the soil by plant roots; however, most of these compounds are applied as post-emergence treatments. Translocation can be in both the xylem and phloem to active growth regions, but their action tends to be localized on the shoot system. They selectively kill broadleaf weeds but can injure grass crops if applied at the wrong

time. In the case of perennial weeds, many of these herbicides translocate to below-ground portions of the plant for systemic kill. Initial symptomology is quickly apparent on newly developing leaves and shoot regions as a twisting and epinasty of the shoot, cupping and crinkling of leaves, elongated leaf strapping (sometimes called "buggy whip") with parallel veins, stem swelling, and a disruption of phloem transport. Secondary effects can be a fusion of brace roots, such as observed with corn. Root injury is expressed as a proliferation or clustering of secondary roots and inhibition of overall root growth. The specific site of herbicide inhibition is not known for this group, although there appear to be multiple sites that disrupt hormone balance, nucleic acid metabolism, and protein synthesis, resulting in alteration of auxin activity in plants, producing weakened cell walls, rapid cell proliferation (unproductive growth), and plant death within several days or weeks.

7. Lipid Biosynthesis Inhibitors (Grass-Specific Herbicides):-

These herbicides have specific activity against grass species only. Dicots and non-grass monocots are tolerant. Some of these herbicides have shown minimal soil activity; however, the main activity occurs after post-emergence application to emerged grass. Activity occurs on both annual and perennial grass species but varies depending on the particular herbicide. Translocation of these herbicides can occur in both the xylem and the phloem, and all generally require the addition of an adjuvant to improve leaf coverage and absorption. These herbicides are most effective when applied to unstressed, rapidly growing grasses. Death of the grass is slow, requiring a week or more for complete kill. Symptoms include rapid cessation of shoot and root growth, pigment changes (purpling or reddening) on the leaves occurring within 2 to 4 days, followed by a progressive necrosis beginning at meristematic regions and spreading over the entire plant. These herbicides inhibit the enzyme acetyl-CoenzymeA carboxylase (ACCase) in the biosynthetic pathway leading to lipid biosynthesis in plants, preventing fatty acid formation, which is essential for plant lipid synthesis. Lack of lipids results in loss of cell integrity of membranes and no new growth.

8. Inhibition of amino acid biosynthesis:-

These herbicides, although differing in chemical structure, all inhibit amino acid synthesis in plants.

1) Group A herbicides inhibit 5-enolpyruvylshikimate-3- phosphate synthase (EPSPS) in the shikimic acid pathway, resulting in limited production of the aromatic amino acids phenylalanine, tryptophan, and tyrosine and many important secondary compounds.
2) Group B herbicides inhibit the enzyme acetolactate synthase [ALS, also called acetohydroxyacid synthase (AHAS)] in the branch chain amino acid pathway, resulting in limited production of isoleucine, leucine, and valine.

These herbicides are potent inhibitors of plant growth and are effective on both dicots and monocots. Glyphosate and sulfosate (Group A) have only foliar activity (no soil activity), and the ALS inhibitors (Group B) have members with foliar, soil, or both soil and foliar activity. Treated plants stop growing almost immediately after application. In the case of EPSPS inhibitors, plants may show a small amount of bleaching around new growth areas; plants die slowly (1 to 2 weeks) and turn a uniform harvest brown color. For ALS inhibitors, 2 to 4 days after treatment, the growing point (apical meristem) becomes chlorotic and later necrotic. Plants may also have shortened internodes, reduced root growth ("bottle brushing"), and pigment changes, including yellowing, purpling, or reddening. Plant death begins at the growing point and gradually spreads to the entire plant, with death occurring within 7 to 10 days.

9. Miscellaneous Herbicides:-

These herbicides inhibit auxin (IAA) transport and/or action in plants, resulting in lack of plant growth due to reduced growth hormone for cell expansion. A common symptom of these herbicides, in addition to reduced plant growth, is the upward turning of the root tip. Such as Naptalam which is soil applied, while diflufenzopyr is foliar applied.

10. Mechanism of Action Not Clear: