Modes of Action of Different Classes of Herbicides

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Abstract

The mode of action of herbicides is important for understanding the management, classification, organization, and hierarchy of the herbicides. It also provides an insight into herbicide resistance, which continues to be a problem in sustainable agricultural management. The overuse of herbicides, just like other pesticides such as insecticides, has led to increased development of resistance among weeds, causing injury and destruction of useful plants in agriculture, land management, and other related industries. This chapter focuses on the main theme while providing in-depth analysis of the different modes of action of various classes of herbicides. The modes of action of herbicides are as variable as their chemical compositions as they focus on controlling susceptible plants through various biochemical means. Depending upon the specific mode of action at work, it may involve a plant enzyme or a biological system that the herbicide may interrupt, thus injuring or disrupting the regular plant growth and development and causing eventual plant death. Having an in-depth knowledge of the mode of action of herbicides is important in choosing a specific herbicide for a specific crop, understanding the injury symptoms, and devising an appropriate crop-management strategy.

Keywords: Herbicides, mode of action, resistance, translocation, regulator

1. Introduction

Herbicides or weedkillers belong to a class of pesticides that are used in the management of undesired plants in the areas of agriculture, landscaping, forestry, gardening, and industry [1, 2]. Weeds cost billions of dollars' worth of damage each year to crops, particularly corn and soybean in the United States and Canada, to which the maximum quantity of herbicides are applied [3-5]. Similar economic and environmental losses have been associated with nonindigenous plant species in Southeast Asia [6,7]. The control of weeds and other unwanted plants in a cost-effective manner is very important to agriculture and other related industries. Herbicide use, though essential for limiting and eliminating the weed populations, poses its own set of problems and risks; its use must be minimized to account for the desired economic and environmental effects [8-10]. The problem associated with weed control is amplified due to herbicide resistance that some of the weed species have developed over the course of time due to the overuse of herbicides or their evolution (process of natural selection) toward favorable conditions [11]. Weeds may be resistant to specific herbicides (selective) or may be resistant to a broad spectrum of herbicides (nonselective) [11-13]. These inherent features have evolved based upon the various mechanisms such as absorption, metabolism, translocation, detoxification, and site of action, which confer resistance to the weeds [14-16].

In order for a successful weed elimination and control strategy to be effective, all of the above conditions must be met. Most of the herbicides, as described in this chapter, interact and interfere with the metabolic machinery and other biochemical pathways of weeds and cause irreversible damage, tissue injury, leading to the eventual death and elimination of the weeds [17]. Herbicides may vary based upon their complex chemical structures, characteristics, and properties with other members of their family, and are grouped according to their mode of action and target specificity [18-20]. The mode of action of herbicides includes inhibition, interruption, disruption, or mitigation of the regular plant growth [21-23].

Herbicides are classified based upon different aspects, such as mode of action, site of action, chemical families, time of application, selectivity, translocation, etc. [24-26]. It is important to note here that even a particular herbicide-resistant weed could be susceptible to a specific herbicide provided the amount and the rate of application are appropriate. On the other hand, excessive use of herbicides could damage the crop and also impart resistance to the same weeds which were intended for control or elimination. Therefore, it is important to strike a balance between these strategies and find the optimum medium for the best and maximum effect. Based upon the time of application, herbicides are classified as preemergence or postemergence [27] as shown in Figure 1. When applied preemergent, they may be effective against grassy weeds or broad-leaf weeds [28,29]. On the other hand, when applied postemergent, they may be selective (specific target) or nonselective (broad target) [28]. At the preemergent stage, the herbicide may be applied to the soil or even the seeds may be treated with them. With postemergent applications, the seedlings are sprayed with specific herbicides so as to eliminate weeds. Selectivity is defined as the capacity of a herbicide to kill a target plant without harming or killing the nontarget plants [30]. Selective herbicides are highly specific and are best suited for the control of a specific weed associated with a specific crop; most of the herbicides used in agriculture and related industries are highly selective. Upon contact, they act by getting absorbed and translocated into the xylem or the phloem of the weeds, by inhibiting or disrupting the metabolic machinery or other biosynthetic pathways, and by injuring or killing the weeds [31]. Nonselective herbicides, on the other hand, have a limited use in agriculture and other related industries, but they are effective in land-reclamation projects where the land needs to be cleared of all vegetation or where the weeds may be localized, away from the plants of interest. Glyphosate, however, has been used worldwide as a nonselective herbicide, but it acts more selectively when used in association with genetically engineered crops, which have been developed for resistance against glyphosate [21,32,33]. The selectivity or nonselectivity of herbicides depends upon various factors, such as plant physiology, soil topography, environment, timing of application, rate of application, and application technique [26]. The classification of herbicides is equally important for managing and understanding herbicide resistance, which continues to be a problem in sustainable agricultural management [24,25]. The overuse of herbicides, just like other pesticides such as insecticides, may lead to increased development of resistance among plants, causing injury and destruction of useful plants in both agriculture and land management [6]. Understanding the reasons for classifying the herbicides based upon their modes of action, instead of the chemical family or the site of action, will help to understand the reasons behind the development of resistance due to their overuse.

Plants interact differently with different herbicides based upon their absorption, translocation, metabolism, and physiological response. The mode of action may be prevalent at the tissue or cellular levels and the tissue-injury symptoms are similar for a specific group. Herbicides are also selective in their mode of action, crop/weed favorability, and soil topology. Herbicides may either be applied directly to the foliage or be added to the soil during plowing/tilling [34]. Herbicides may have a vertical or horizontal translocation movement of the chemicals representing different groups [35-37]. There are other herbicides that kill upon contact on the foliage and are potent enough such that they do not require translocation either way. Plants are intact systems that consist of organs, tissues, cells, and molecules, which are reservoirs of organized biochemical processes that take place uninterrupted. Herbicides may be absorbed by the plants via the roots (soil-based herbicides) or the shoots (spray-based herbicides) [38]. The metabolic activity requires the movement of sap through the xylem (translocation of water and nutrients) and phloem (translocation of sugars) [39,40]. When the herbicides penetrate the cell walls of the weeds, they cause tissue injury and permeate the sap, in the process, interrupting various biochemical pathways. Upon interaction with the herbicides, weeds are killed by the dysfunction of their biochemical processes.

Traditionally, herbicides were mixed with the soils, but at present, the trend is leaning toward spraying herbicides and also developing herbicide-resistant crop varieties [6,41]. Given the environmental concerns associated with aerosols, soil pollution, and water-system contamination, developing environment-friendly herbicides has become a priority [42,43]. The United States Environmental Protection Agency (US EPA) has many regulations and guidelines in place for the proper manufacturing, sale, and use of herbicides (www.epa.com). The rate of application of herbicides and their strategic placement are of prime importance and depend upon the kind of weeds that need to be killed. Herbicides with higher rates of absorption and retention (during spraying) require a less volume and a less potency as compared to their counterparts. The weather/temperature conditions (mild, temperate, or tropical) determine the effectiveness of herbicides on a specific crop. Along with the temperature, the humidity and the plant vigor also play important roles in designing the herbicide application strategy [44,45]. An understanding of the leaf-surface coverage area, leaf-surface properties, and the chemical properties of the herbicide is essential for maximum success [46-48].

Herbicides are grouped based upon their chemical structures, which consist of a base-specific molecule surrounded by a side chain or a group(s) [49]. A modification of a functional group leads to a modification in the activity, selectivity, and persistence of herbicide and also

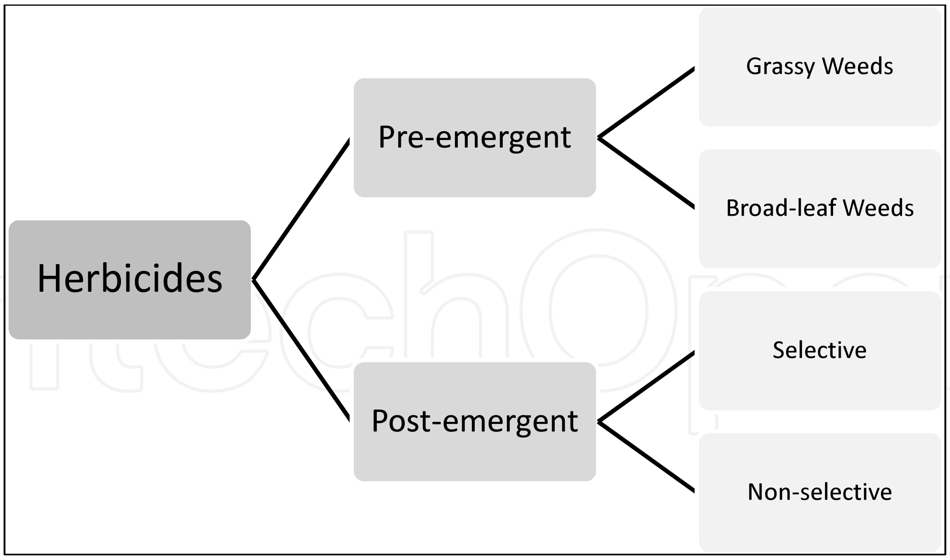

Figure 1. Herbicide classification and application

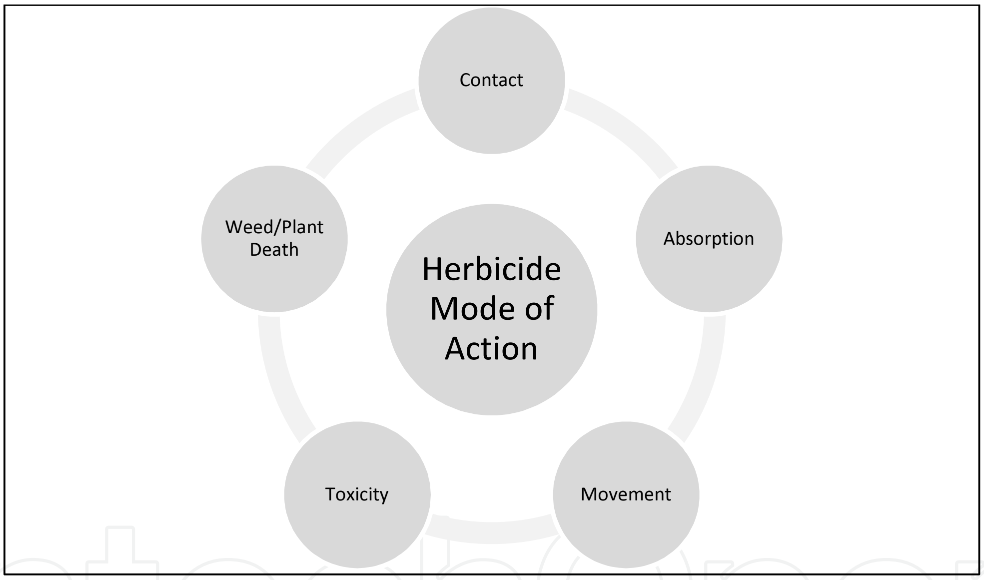

Figure 2. Herbicide mode of action – stages

determines its mode of action. The overuse of herbicides has increased the chances of damage and injury to the non-target weeds/plants while also causing groundwater contamination [6,10,50). This chapter describes the different modes of action of herbicides based upon whether they are inhibitors, regulators, or disruptors of various biosynthetic pathways and hence classified accordingly. Slight variations have been observed in the group-classification system in scientific publications but those are of minor concern. The mechanism of the action of herbicides for killing a weed is well understood and requires that, in order for it to be effective, the herbicide must undergo the following processes, in the sequential order, listed below [51] and also as illustrated in Figure 2:

1. Contact – Must come in contact with the weed
2. Absorption – Must be absorbed by the weed
3. Movement – Must move to the site of action in the weed
4. Toxicity – Must possess or acquire sufficient level of toxicity or potency to kill the weed
5. Death – Must cause injury to the weed leading to eventual death

2. Herbicide resistance

Herbicide resistance is a widespread problem in agriculture and related industries [11,21]. The resulting loss of crops can be significant for farmers and other growers. Instead of using a passive approach to herbicide-resistance management, the use of proactive strategies based upon the latest advancements and knowledge will deliver the best results [52-54]. The results of a successful herbicide-resistance management strategy can be quantified in terms of both the cost-effectiveness and the total output of the harvest [55]. The maximum loss occurs during the initial stage of the crop's planting, particularly when the soil and/or the seeds are not treated with herbicides [56,57]. It is at this stage that the seeds are the most vulnerable and the weeds are the most robust and must be treated accordingly [34,58]. If left untreated or not appropriately treated, the weeds can get out of control, take over the crop/plants, and also become resistant leading to long-term and devastating economic consequences for farmers and growers [59,60]. Therefore, it is important to select a weed-resistant variety of crop instead of attempting to deal with the populations of weeds, which may be resistant to such herbicides, thus increasing the overall cost of cultivation [61,62].

Weeds have been shown to have acquired unprecedented resistance to several herbicides due to their indiscriminate use over the years [21,63]. This may be due to the result of mutation or substitution of a single base, thus changing the reading frame of the amino acid and the amino acid itself [64-68]. This modification, usually in the quinone-binding region of the peptide, decreases the binding capacity of the herbicide and renders it to be vulnerable and less effective [49,69,70]. If the mutation is at the gene level, it may be a single-gene mutation or a multigene mutation and would impart the weed a higher or lower level of resistance, respectively [71-74]. Recent advances have shown that slight alterations in the binding affinity of the herbicides to the crops, via different pathways, particularly the photosynthetic pathway, have led to the development of various effective herbicide strategies [69,75,76]. Despite the concerns of genetically modified (GM) crops, the recombinant DNA technology is one such tool that allows for the development of crop-safe and effective herbicides, which kill only the weeds when applied to the entire crop [77,78]. However, the processes of natural selection and gene transfer may allow the weeds to acquire herbicide resistance quickly [21,79]. On the other hand, identifying resistance among weeds is an extremely challenging task, which requires different tools at one's disposal [12]. Some of the observational tools, necessary for controlling the weed and other undesirable plants during cultivation, include:

1. Loss of consistent control of the weed population

2. Effect of environment/weather conditions on the weed population
3. Rate of application of herbicides on the weed population
4. Soil topography (effect of overuse or crop rotation on the weed population)
5. Use of herbicides with a similar mode of action
6. Use of herbicides with related chemical families

Herbicide resistance, like antibiotic resistance, is a common occurrence among weeds and is a result of overuse of a specific herbicide and soil conditions [21,80]. In order to avoid and overcome the development of herbicide resistance, different herbicides must be used at different points in time on the same crop [81]. Crop rotation is also recommended to maintain soil integrity and soil vigor [29]. The genotype of different plants lends them to interact differently due to conformational variations and compatibility with various chemical groups [82]. For example, Kochia and pigweed are resistant to triazine and ALS-inhibiting herbicides [83]. Certain genetically modified (GM) crop plants, which may inadvertently come in the path of the herbicides (carry-over) may able to overcome the harmful effects of these herbicides [11,84,85]. Cross-resistance is another concerning issue in agriculture and related industries as some weeds and pests have acquired resistance to several herbicides, which are related by their mode of action [86-88]. For example, herbicides belonging to separate chemical families but within the same mode of action [e.g., acetolactate synthase (ALS) inhibitors] may acquire herbicide resistance against both the chemical families. Crop rotation on the same piece of land is highly influential and beneficial in minimizing the development of herbicide resistance as the soil comes in contact with various microorganisms and interacts differently with different chemicals in the surrounding micro-ecosystem [21,29]. The crop rotation allows for different herbicides to be used, which reduces the selection for resistance. The weed-control effect is the maximum at the emergence stage and the minimum at the maturity stage, as the plant goes through the stages of emergence, seedling, vegetative, flowering, and maturity, respectively [89].

3. Mode of action

The modes of action of herbicides are as variable as their chemical compositions and focus on controlling susceptible plants through various biochemical means. The weed-control effect is the maximum at the emergence stage and the minimum at the maturity stage, as the plant goes through the stages of emergence, seedling, vegetative, flowering, and maturity, respectively [89]. Depending upon the specific mode of action at work, it may involve a plant enzyme or a biological system that the herbicide may interrupt, thus injuring or disrupting the regular plant growth and development and causing eventual plant death. Extensive research in the area of herbicides has led to their classification based upon their modes of action into various groups, which are discussed in detail in this chapter. It is important to note that the modes of action discussed in this chapter, though comprehensive, are not exhaustive by any measure. Newly discovered groups and unexplained (unknown) groups continue to be added to the list, reflecting slight variations of classification, as new research knowledge continues to emerge. Also, some scientists may have a slightly different method of classification as well as different

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groups, but those are only slight variations. What is important, however, is that having an in-depth knowledge of the mode of action of herbicides is necessary in choosing a specific herbicide for a specific crop, understanding the injury symptoms, and devising an appropriate crop-management strategy and using an appropriate herbicide. Even though this chapter focuses on the herbicide's mode of action, it also introduces the reader to a vast array of topics such as herbicides, herbicide resistance, group-numbering system for designating various herbicides based upon their mode of action, etc. Herbicides belonging to a specific group have the same mode of action even though they may belong to a different chemical family. Finally, the environmental fate of herbicides (persistence, degradation, mobility) with specific reference to the groundwater, water sterilization, soil contamination, and environmental and public health concerns will enlighten the reader on the importance of using the herbicides diligently and according to the regulations, guidelines, and labeling. The modes of action, as discussed in this chapter, are listed below:

1. Lipid biosynthesis inhibitors
2. Amino acid biosynthesis inhibitors
3. Plant growth regulators
4. Photosynthesis inhibitors
5. Nitrogen-metabolism inhibitors
6. Pigment inhibitors
7. Cell-membrane disruptors
8. Seedling-growth inhibitors

3.1. Group 1: Acetyl Coenzyme A Carboxylase (ACCase) inhibitors

Also known as lipid biosynthesis inhibitors, these herbicides inhibit the ACCase enzyme activity and are used typically for controlling grass during the cultivation of broadleaf crop varieties or crop rotation. The ACCase enzyme catalyzes the primary step in the fatty-acid synthesis, thus blocking the production of phospholipids necessary for synthesizing the lipid bilayer, which is indispensable for cell structure and function [90-92]. The chemical family of aryloxyphenoxypropionate, cyclohexanedione, and phenylpyrazolin operates by inhibiting the ACCase enzyme. These herbicides are also known by their chemical family nicknames – FOPs, DIMs, and DENs [93]. Many broadleaf crop varieties, including grasses, have a natural resistance to these herbicides due to a strong and less-sensitive ACCase system [94].

3.2. Group 2: Acetolactate Synthase (ALS) inhibitors

Also known as amino acid synthesis inhibitors, these herbicides inhibit the action of the acetolactate synthase (ALS) enzyme. Also known as acetohydroxy acid synthase (AHAS), ALS catalyzes the first step in the synthesis of the branched-chain amino acids, such as leucine, isoleucine, and valine [95]. These are also referred to as the AHAS inhibitors or branched-chain amino acid inhibitors. Comprising the imidazolinones, pyrimidinylthiobenzoates, sulfonylaminocarbonyltriazolinones, sulfonylureas, and triazolopyrimidines chemical family, the ALS inhibitors are a part of the largest group of herbicides, which function with the amino acid synthesis inhibitor mode of action [96]. They may show cross-resistance to other herbicides and act by reducing the production of the branched amino acids in the presence of the ALS enzyme, causing plant wilting and, ultimately, plant death.

3.3. Group 3: Root growth inhibitors

Also known as the seedling root growth inhibitors, these herbicides inhibit cell division as part of their mode of action, which, ultimately, blocks root extension and growth. They are applied preemergent or preplant in vegetables and ornamentals. Their site of action is the microtubule and is marked by the assembly of the herbicide–tubulin complex inside the microtubules. This complex inhibits the polymerization of microtubules during assembly but remains unaffected during depolymerization [97]. A loss in the structure and function of the microtubule causes cell death by compromising the cell-wall formation due to the nonalignment of the spindle fibers and nonseparation of the chromosomes during mitotic cell division. This group is represented by the benzamide, benzoic acid [dimethyl-2,3,5,6-tetrachloroterephthalate (DCPA), dinitroaniline, phosphoramidate, and pyridine chemical family, which act by causing the loss of microtubule formation leading to the obstruction of cell division and elongation as evidenced by the swelling of the root tips.

3.4. Group 4: Plant growth regulators

Also known as synthetic auxins, this group includes hormone-based herbicides and is used to keep broadleaf weeds out during the cultivation of corn, wheat, and sorghum. The mode of action of the endogenous indole acetic acid (IAA) is mimicked by the herbicides belonging to the chemical family represented by benzoic acid, phenoxycarboxylic acid, pyridine carboxylic acid, and quinoline carboxylic acid [98]. The specific molecular binding site responsible for the IAA activation is yet to be established and remains unknown. All of these chemicals disrupt the nucleic acid metabolism and the cell-wall integrity by activating the adenosine triphosphate (ATP)ase proton pump, which increases the enzyme activity in the cell wall [99]. These regulators mimic the IAA activity, thus increasing the transcription, translation, and the protein biosynthesis activities within the cell leading to uninhibited vascular growth, causing cell bursts and ultimate cell and plant death.

3.5. Groups 5, 6, and 7: Photosynthesis inhibitors – Photosystem II (PSII) inhibitors

The mode of action of these herbicides is the inhibition of the photosynthetic pathway, specifically the Photosystem II (PSII). Due to their excessive use, some weeds have become resistant to these herbicides developed on this metabolic principle. Group 5 is represented by the chemical family of triazine, triazinone, phenylcarbamates, pyridazinones, and uracils. Group 6 is exemplified by nitriles, benzothiadiazinones, and phenylpyridazines. Group 7 comprises phenyl urea and amides. All of these groups represent different binding schemes as compared to each other with several similarities. All of these PSII herbicides inhibit the photosynthetic pathway by binding the Q_B-binding site of the D1 protein complex present in the chloroplast thylakoid membrane. The binding disrupts the electron transport system (ETS) from Q_A to Q_B and also blocks the CO_2 fixation, ATP generation, and nicotinamide adenine dinucleotide hydrogen phosphate ($NADPH_2$) production required for various biochemical pathways as part of the plant growth and development [70,100]. As a result of the blockage at the level of the electron transport chain (ETC), the plant is unable to reoxidize Q_A, which generates the triplet-chlorophyll (^3Chl), which forms singlet-oxygen ($^1O_2$) upon reacting with molecular oxygen (O_2). The unsaturated fatty acids and lipids release hydrogen in the presence of triplet-chlorophyll (^3Chl) and singlet-oxygen ($^1O_2$) while forming a lipid radical, thus causing lipid peroxidation. Lipid peroxidation causes the lipids in the bilayer and other proteins to be oxidized, producing reactive oxygen species (ROS) [70,100]. Some of these herbicides also cause the disruption of the carotenoid, anthocyanin, and protein biosynthetic pathways and affect the transcription machinery as well. The oxidation, ultimately, causes the loss of chlorophyll and other pigments like the carotenoids from the cell membranes exposing the cells and cell organelles to harsh conditions leading to their collapse, disintegration, and eventual plant death [101,102]. Due to their overuse, some weeds have acquired resistance to these PSII inhibitor herbicides, such as atrazine and metribuzin [103].

3.6. Groups 8 and 15: Shoot-growth inhibitors

Also known as seedling shoot growth inhibitors, the herbicides designed with this mode of action are applied as part of the soil preparation and act effectively before the grass and broadleaf weeds emerge. The site of action of the Group 8 herbicides is at the location of the lipid synthesis machinery in the cell membrane. These Group 8 herbicides are represented by the chemical family of phosphorodithioates and thiocarbamates and inhibit the biosynthesis of lipids, fatty acids, proteins, isoprenoids, flavonoids, and gibberellins [104]. The site of action of the Group 15 is at the very-long-chain fatty acid (VLCFA) location in the cell membrane [105,106]. Group 15 herbicides are represented by the chemical family of chloroacetamide, acetamide, oxyacetamide, and tetrazolinone. These herbicides conjugate with acetyl COA and certain sulfhydryl-containing molecules via thiocarbamate sulfoxides, which inhibit the long-chain fatty acids during the seedling shoot growth stage of the plant and affect the weeds' preemergence.

3.7. Group 9: Aromatic amino acid inhibitors

The mode of action of these herbicides is as an amino acid synthesis inhibitor. This mode of action is specific to glyphosate (glycines), which are nonspecific herbicides that act by inhibiting the amino acid synthesis. These herbicides kill or cause injury to any plant that they come in contact with and hence are approved only for use in glyphosate-resistant crops, such as corn, cotton, canola, and soybean. Glyphosates inhibit the 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase enzyme necessary in the generation of the EPSP from shikimate-3-phosphate and phosphoenolpyruvate as part of the shikimic acid pathway [107]. As a result, the necessary levels of the aromatic amino acids (tryptophan, tyrosine, and phenylalanine) are depleted, thus compromising the biosynthetic metabolic pathways leading to eventual plant death [108]. As a herbicide, glyphosate use is the most prevalent in the world due to its broad-spectrum properties and for being environmentally- and user-friendly. Glyphosates are available as ammonium salts, diammonium salts, dimethylammonium salts, isopropylamine salts, as well as potassium salts. The ease of access to the target (broad-spectrum), and the ease of translocation (via the xylem and the phloem), and the failure of the weeds to overcome their effect make the Group 9 herbicides as ideal candidates for killing weeds associated with the glyphosate-resistant crops.

3.8. Group 10: Glutamine-synthesis inhibitors

The mode of action is nitrogen metabolism-based and is specific to glufosinate, which is nonspecific in nature. These herbicides can be used in glufosinate-resistant crop cultivation during postemergence of the resistant seedlings. Glufosinate and bialophos (phosphinic acids) inhibit the activity of the enzyme, glutamine synthetase (GS), which converts glutamate and ammonia to glutamine [109,110]. GS plays a key role in nitrogen metabolism (nitrogen fixation and nitrate/ammonia nutrition) by re-assimilating the ammonia generated during respiration. These herbicides disintegrate the proteins by disrupting the optimum activity of GS leading to an accumulation of ammonia, which lowers the pH gradient on either sides of the cell membrane. This causes the disruption of various cell functions, particularly shutting down the PSI and PSII systems leading to the uncoupling of the photophosphorylation [100,111]. Since GS is located in important organelles like the chloroplast and cytoplasm, these herbicides are highly effective in controlling weeds and other undesired plants.

3.9. Groups 12, 13, and 27: Pigment synthesis inhibitors

Also known as carotenoid biosynthesis inhibitors, these herbicides destroy the green pigment, chlorophyll, which is necessary for photosynthesis in the plants. These herbicides are also known as bleachers as they impart a white color to the plant tissues after coming in contact with the plant foliage leading to cell and tissue injury and ultimately killing the weeds. As part of their mode of action, these herbicides inhibit the pigment synthesis, specifically the catalysis of the 4-hydroxyphenyl pyruvate dioxygenase (HPPD) enzyme and are also referred to as the HPPD-inhibitors. Group 12 is represented by the chemical family of amides, anilidex, furanones, phenoxybutan-amides, pyridiazinones, and pyridines. These herbicides containing these chemicals disrupt the carotenoid biosynthetic pathway by inhibiting the function of the phytoene desaturase enzyme [112]. Group 13 is represented by the chemical family, Isoxazolidinone, and the site of action is at the location of the diterpene synthesis. Group 27 is represented by the chemical family, Isoxazole, and they are also HPPD inhibitors. Carotenoids play a key role in quenching the oxidative control of singlet O_2 ($^1O_2$) among healthy plants. Upon being treated with these herbicides containing the pigment synthesis inhibitors (Groups 12, 13, and 27), the level of carotenoids is highly reduced leading to the presence of unbound lipid radicals. These lipid radicals compromise the uptake of the membrane lipids and fatty acids, causing lipid peroxidation, which renders the chlorophyll, other cell membrane lipids, and some proteins dysfunctional. As a result of membrane leakage, the cell contents are exposed and destroyed rather rapidly causing wilting and eventual plant death.

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3.10. Group 14: PPO inhibitors

The PPO inhibitors act by disrupting the cell membranes and hence their mode of action is categorized as cell membrane disrupters and their site of action is the cell membrane. Most of these herbicides are applied at the postemergence stage while some are used at the preemergence stage of the seedling. The mode of action of these herbicides depends upon the inhibition of protoporphyrinogen oxidase (PPO) enzyme (also known as PPG oxidase or Protox inhibitors), which catalyzes the chlorophyll and heme biosynthesis [protoporphyrinogen IX (PPGIX) to protoporphyrin IX (PPIX) oxidation catalysis]. These herbicides are represented by the chemical family, diphenylether, aryl triazolinone, *N*-phenylphthalimides, oxadiazoles, oxazolidinediones, phenylpyrazoles, pyrimidindiones, and thiadiazoles. As the PPO is inhibited, it causes the over-accumulation of the PPIX, which interacts with the light in the chlorophyll and produces the triplet-PPIX. Upon reacting with O_2, triplet-PPIX forms $^1O_2$ causing the hydrogen-bond disruption in the unsaturated fatty acids and lipids in the membrane, which causes lipid peroxidation [113]. As a result of the cell membrane disruption, the lipids and proteins become oxidized causing the chlorophyll and other pigments to leak and causing cell disintegration, wilting, and eventual plant death.

3.11. Group 22: Photosynthesis inhibitors – Photosystem I (PSI) inhibitors

Upon contact with the plant foliage, these herbicides act by penetrating and destroying the cell lipid bilayer leading to the breakdown of the cell membranes and hence their mode of action is categorized as cell membrane disrupters. These herbicides are nonselective in nature and are applied usually prior to harvesting the crop. These herbicides are represented by the bipyridilium chemical family and are also known as PSI electron diverters as they accept electrons from PSI and, in the process, generate herbicide radicals. Upon interacting with O_2, the herbicide radicals form superoxide radicals, which, in the presence of the superoxide dismutase enzyme, form hydrogen peroxide (H_2O_2) and hydroxyl radicals [114]. These radicals disrupt the unsaturated fatty acids, chlorophyll, lipids, and proteins in the cell membrane. As a result, the cell membrane is disrupted beyond repair causing leakage of the cell cytoplasm, which leads to wilting and eventual plant death.

4. Conclusion

Due to the overuse of herbicides, agricultural weeds and other undesirable plants develop resistance, which must be contained or eliminated in order for the maximum output of the harvest. Herbicide resistance occurs due to the overuse of herbicides over the years. It is important to follow the lead in developing a proactive and robust herbicide resistance management strategy for minimizing the agricultural loss. Similar techniques can be extrapolated to the land management, ornamental, and other related industries to minimize the evolution of the herbicide-resistant varieties of weeds and other pests. Based upon the available body of knowledge, recent advances in agricultural research, and latest techniques, it is advisable to use different herbicides (different modes of action) at different times of the year and with different crops so that the weeds do not develop resistance to the herbicides as quickly as that have been reported. Deciding which herbicide is the most effective and the most environmental friendly option for a specific crop can be a daunting task particularly with so many products competing for attention in the multibillion dollar herbicides market. Most of these herbicides are developed based upon similar weed control and pest management strategies and are designed based upon their mode of action. Rearranging various chemical groups within the chemical family, which most of the companies tend to do while developing their key products, may not be the best strategy as the weeds tend to develop a quick resistance to such chemicals or groups of chemicals. Therefore, it is imperative that the herbicides be designed to obtain the maximum effect with regard to their mode of action so as to control or eliminate weeds and destroy their capacity to acquire the herbicide resistance.

Group	Mode of Action	Site of Action	Chemical Family
1	Lipid-Synthesis Inhibitors	ACCase Inhibitor	Aryloxyphenoxypropionate (FOPs), Cyclohexanedione (DIMs), Phenylpyrazolin (DENs)
2	Amino-Acid Synthesis Inhibitors	ALS Inhibitors	Imidazolinones, pyrimidinylthiobenzoates, sulfonylaminocarbonyltriazolinones, sulfonylureas, triazolopyrimidines
3	Root-Growth Inhibitors	Microtubule Inhibitors	Benzamide, benzoic acid (DCPA), dinitroaniline, phosphoramidate, pyridine
4	Plant-Growth Inhibitors	Site of Action Unknown	Benzoic acid, phenoxycarboxylic acid, pyridine carboxylic acid, and quinoline carboxylic acid
5	Photosynthesis Inhibitors	Photosystem II Inhibitors	Triazine, triazinone, phenylcarbamates, pyridazinones, and uracils.
6	Photosynthesis Inhibitors	Photosystem II Inhibitors	Nitriles, benzothiadiazinones, and phenylpyridazines
7	Photosynthesis Inhibitors	Photosystem II Inhibitors	Phenyl, urea, and amides
8	Shoot-Growth Inhibitors	Lipid-Synthesis Inhibitors	Phosphorodithioates and thiocarbamates
9	Amino-Acid Synthesis Inhibitors	EPSP Synthase Inhibitors	Not designated by any specific chemical family
10	Nitrogen-Metabolism Inhibitors	Glutamine-Synthesis Inhibitors	Not designated by any specific chemical family
12	Pigment-Synthesis Inhibitors	HPPD Inhibitors	Amides, anilidex, furanones, phenoxybutan-amides, pyridiazinones, and pyridines
13	Pigment-Synthesis Inhibitors	Diterpene-Synthesis Inhibitors	Isoxazolidinone
14	Cell-Membrane Disrupters	PPO Inhibitors	Diphenylether, aryl triazolinone, *N*-phenylphthalimides, oxadiazoles,

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Group	Mode of Action	Site of Action	Chemical Family
			oxazolidinediones, phenylpyrazoles, pyrimidindiones, and thiadiazoles.
15	Shoot-Growth Inhibitors	Very-Long-Chain Fatty Acid (VLCFA) Inhibitors	Chloroacetamide, acetamide, oxyacetamide, and tetrazolinone.
22	Cell-Membrane Disrupters	PSI Inhibitor	Bipyridilium
27	Pigment-Synthesis Inhibitors	HPPD Inhibitors	Isoxazole

Table 1. Modes of action of different classes of herbicides

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