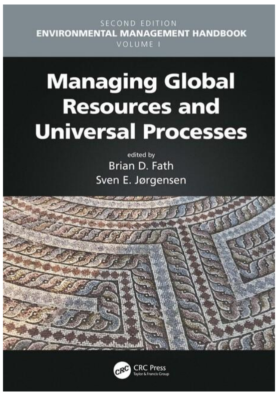

Managing Global Resources and Universal Processes

Brian D. Fath, Sven E. Jørgensen, Megan Cole

Herbicides

Publication details

https://www.routledgehandbooks.com/doi/10.1201/9780429346132-4

Malcolm Devine

How to cite :- Malcolm Devine. 30 Jul 2020, *Herbicides from:* Managing Global Resources and Universal Processes CRC Press
Accessed on: 19 Jun 2021
https://www.routledgehandbooks.com/doi/10.1201/9780429346132-4

3

Herbicides

Malcolm Devine

Herbicide Discovery

Traditionally, herbicides have been discovered through large chemical synthesis and screening programs. Given the high cost of discovery and development of new compounds, efforts have focused on herbicides that target major weeds in major world crops (e.g., corn, rice, wheat, soybean, cotton). However, these compounds often find uses in minor crops, also. The process normally involves a step wise progression from greenhouse screening on a few crop and weed species, to more extensive indoor testing, and eventually to field trials in many locations examining the interactions between different weed species, soil types, weather conditions, etc. Toxicological testing and formulation improvement proceed concurrently, to ensure that regulatory and efficacy requirements are met.

A recent innovation is the use of combinatorial chemistry to identify lead compounds. Rather than being synthesized and evaluated in isolation, compounds are produced and screened as mixtures. When combined with in vitro screens (activity testing at the biochemical or cellular level) rather than whole-plant assays, this can allow the testing of 20- to 50-fold more compounds per year than a traditional herbicide discovery program.

"Rational" discovery of herbicides involves identification of a candidate herbicide target site in the plant, followed by design of inhibitors that specifically block that target. While this has led to the discovery of some novel enzyme inhibitors, to date no commercial herbicides have been discovered through this approach. One difficulty is that compounds predicted to have high activity might not penetrate the tissue satisfactorily or may be rapidly degraded inside the tissue.

Finally, herbicides can be developed from bacterial, fungal, or plant toxins. One commercial herbicide, glufosinate (= phosphinothricin), was developed from the bacterial toxin bialaphos from *Streptomyces hygroscopicus.*

Herbicide Classification

Method or Timing of Application

Preplant incorporated herbicides are applied to the soil surface and mechanically incorporated into the upper 5–10 cm of the soil, in order to minimize photodecomposition and volatilization losses. Preemergence herbicides are applied to the soil surface and often rely on precipitation or soil moisture to transport them to the plant root or shoot for uptake. Postemergence herbicides are applied to exposed foliage after the plants have emerged.

Chemical Structure or Mode of Action

Herbicides within the same structural family usually have the same mode of action, with varying degrees of activity or selectivity depending on the structural variations between compounds. However, herbicides from different chemical families can have the same mode of action (see later).

Components of Herbicide Action

To be effective, herbicides must penetrate the tissue and reach the target site in sufficiently high concentrations to block its activity. The overall process of herbicide action can be separated into the following components:[1]

Absorption: Herbicides can be absorbed by the roots or directly into the leaves. Root uptake occurs through mass flow of herbicide in soil moisture and is a function of root distribution in the soil, soil moisture status, the physical properties of the soil, and the behavior of the herbicide in the soil. Foliar absorption occurs following application to the leaves. To facilitate entry through the cuticle (the waxy layer on most leaf surfaces), herbicides are usually formulated with an array of inert ingredients including surfactants, emulsifiers, etc. Once inside the tissue, further penetration through cell walls and membranes usually occurs by simple diffusion.

Translocation: Long-distance transport of herbicides in the plant can occur in the xylem and/or phloem. In some instances, distribution through the plant is a critical component of overall activity. The amount of translocation depends on the plant stage of development, the physicochemical properties of the herbicide, and the rate at which herbicide injury slows down the movement of endogenous compounds.

Metabolic degradation: Plants have evolved various enzyme systems to detoxify potentially harmful compounds. Fortuitously, some of these enzymes (e.g., cytochrome P450 monooxygenases, glutathione S-transferases) can degrade herbicides to inactive compounds.

Interaction at the target site: Finally, all herbicides must interfere with some critical process in the plant. In most cases this involves binding to a protein (usually a functional enzyme, or a transport or structural protein) so that it cannot carry out its normal function. Over time, through the combined effect of this direct action and other indirect actions, the plant dies.

Herbicide Selectivity

Most herbicides are selective, that is, they kill some plant species but not others. A few herbicides, on the other hand, are nonselective, and kill essentially all species. Selectivity is normally based on one of the following:[1]

- Failure to absorb the herbicide, due to either selective placement (e.g., directed spray on weeds growing between the crop rows) or failure of the herbicide to reach the roots of deep rooted crops, while killing shallow rooted weeds.

- Enhanced rate of metabolic degradation of the herbicide in the crop. This is the most common basis of selectivity of agricultural herbicides. After entry into the plants, the crop metabolizes the herbicide to inactive compounds, whereas degradation does not occur or is slower in the susceptible weeds.
- Differential sensitivity of the target site. This is particularly common in the case of herbicide resistant weeds (see later).

Herbicide Mode of Action

Herbicides kill plants by interfering with an essential process in the plant. The major modes of action of herbicides, the biochemical target sites, and some examples of chemical groups that interfere with those targets, are shown in Table 1.

In addition, many herbicides have been identified that interact with other, unique target sites in plants. However, most of the herbicides that have been developed over the past 50 years target about 15 distinct molecular targets.

Herbicide Resistance in Weeds

The repeated use of herbicides can lead to the development of herbicide-resistant weed populations. The use of herbicides per se does not create herbicide resistance. Rather, the continuous selection pressure through herbicide use creates a niche, allowing resistant individuals to survive and increase in

TABLE 1 Modes of Action of Major Herbicide Groups[a]

Mode of Action	Target Site	Representative Chemical Groups
	Inhibition of amino acid biosynthesis	
Branched chain amino acids	Acetolactate synthase	Sulfonylureas, imidazolinones, triazolopyrimidines
Glutamine synthesis	Glutamine synthetase	Glufosinate
Aromatic amino acid biosynthesis	Enolpyruvylshikimate phosphate synthase	Glyphosate
	Photosynthesis	
Photosynthetic electron transport (PS II)	Q_b or D1 protein	S-Triazines, phenylureas, benzonitriles
Photosynthetic electron transport (PS I)	PS I electron acceptor	Bipyridiliums
	Pigment biosynthesis	
Chlorophyll synthesis	Protoporphyrinogen oxidase	(Nitro) diphenylethers
Carotenoid synthesis	Phytoene desaturase and others	Aminotriazole, clomazone
	Lipid biosynthesis	
Fatty acid synthesis	Acetyl-coA carboxylase	Aryloxyphenoxypropionates, cyclohexanediones
Fatty acid elongation	"Elongase" complex	Thiocarbamates
	Cell division	
Spindle formation	β-Tubulin	Dinitroanilines, carbamates
	Other	
Auxin disruption	Auxin binding proteins (?)	Phenoxyacetic acids, benzoic acids
Homogentisate biosynthesis	4 hydroxyphenylpyruvate dioxygenase	Isoxazoles

[a] From Devine.[1]

the population as susceptible weeds are killed. Thus, starting with an initial population of perhaps one resistant weed in a population of 10^6–10^9, resistance builds up over time until the resistant weeds become predominant in the field.

Herbicide resistance in weeds was first observed in the late 1950s, but was a minor problem until the mid-1970s, when resistance to triazine herbicides became a widespread concern. Since then the occurrence of resistance has increased dramatically, with >200 cases now reported.[2]

In most cases, resistance is due to a point mutation in the gene coding for the herbicide target site.[3] This alters the structure of the target protein in such a way that it can still perform its natural function, but herbicide binding is reduced. This type of resistance often confers cross-resistance to herbicides in the same chemical family or mode-of-action group, although exceptions exist and each case must be analyzed separately. However, target-site mutations do not alter the sensitivity of the weed to herbicides with other mechanisms of action.

Resistance can also be conferred by elevated activity of the enzyme(s) responsible for herbicide degradation. These weeds can be cross-resistant to other herbicides with different mechanisms of action. Again, the possibilities for cross-resistance have to be analyzed on a case-by case basis.

Although herbicide resistance has become widespread, in almost all cases alternative control methods are available, through the use of other herbicides, changes in cropping or tillage practices, or some combination of these. Avoidance and management of herbicide resistance has become an integral part of good farming practices in modern agriculture.

Herbicide-Resistant Crops

A recent development in selective weed control has been to create resistance to certain herbicides in crops where it did not exist previously. While this has been done primarily to extend the market share of certain products, it offers farmers the advantage of broad-spectrum weed control in crops with a single herbicide application.[4] In some cases this has substantially reduced the total amount of herbicide required in a single season.

Herbicide-resistant crops can be produced by three methods:[4]

1. Making crosses between the crop (sensitive to the herbicide) and a related, resistant species. This method was used to develop triazine-tolerant canola (*Brassica napus*).
2. Selecting resistant cells in tissue culture, through random mutation or by selecting somaclonal variants, and regenerating resistant plants from these cells. Corn lines resistant to the herbicide sethoxydim were generated in this way.
3. Transfer of an herbicide-resistance gene through genetic engineering. The gene is identified in an unrelated species (often a bacterium), cloned, and transferred into the crop species of interest. These procedures were used to develop canola varieties resistant to the nonselective herbicides glufosinate and glyphosate.

Herbicide-resistant crops have greatly facilitated weed control, but present some new research questions that have had to be addressed. These include the likelihood and long term ecological consequences of gene flow to related species, and the need to control volunteer plants in the following season(s). These issues do not present insurmountable obstacles but, again, need to be dealt with on a case-by-case basis.

Safety and Environmental Fate of Herbicides

Environmental safety is of prime concern in the development of new herbicides. This includes an understanding of herbicide toxicology, safe handling and application procedures, and environmental behavior and fate. In most countries, approval of herbicides by the relevant decision making bodies is dependent on the registrant satisfying the regulatory requirements imposed by those countries.

Toxicological requirements vary from country to country, but usually include data on oral and dermal toxicity in a range of species, in tests of varying duration. Based on the data collected, maximum residue levels are established in food or food products. Data from these tests and field experiments are used to establish maximum application doses and safe intervals between product application and harvest or grazing. Data may also be required on effects on nontarget organisms and ecosystems that may be exposed to low herbicide doses.

Appropriate handling procedures are an important aspect of herbicide safety. The use of appropriate safety clothing (gloves, coveralls, masks, etc.), more benign formulations (e.g., dispersible granules rather than wettable powders), etc., contribute to reduced applicator exposure. Recently, novel formulations have been developed that further reduce applicator exposure when adding products to the spray tank.

Herbicide drift immediately after spraying can be a source of off-site contamination, resulting in injury to adjacent sensitive crops and other species. Various measures, including spraying only under calm conditions, use of wind deflectors, and avoiding very small droplets, can substantially reduce the risk of spray drift.

Herbicides in soil are lost by a combination of microbial and chemical degradation, plant uptake, and, in some instances, leaching or surface run-off. Stringent environmental regulations have been introduced in many countries to minimize the possibility of groundwater contamination. Herbicide residues in soil can provide extended weed control, but also may limit crop rotation options in future seasons. Field research is conducted to establish the risk of such carryover and the effects on future crops.

References

1. Devine, M.D.; Duke, S.O.; Fedtke, C. *Physiology of Herbicide Action*; Prentice-Hall: Englewood Cliffs, NJ, 1993; 441.
2. Heap, I.M. International survey of herbicide-resistant weeds. http://www.weedscience.com (accessed April 2000).
3. Devine, M.D.; Eberlein, C.V. Physiological, Biochemical and Molecular Aspects of Herbicide Resistance Based on Altered Target Sites. In *Herbicide Activity: Toxicology, Biochemistry and Molecular Biology*; Roe, R.M., Burton, J.D., Kuhr, R.J., Eds.; IOS Press: Amsterdam, 1997; 159–185.
4. *Herbicide-Resistant Crops. Agricultural, Environmental, Economic, Regulatory and Technical Aspects*; Duke, S.O., Ed.; CRC Press: Boca Raton, FL, 1996; 420.