Chapter 8: Weed Resistance to Herbicides

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Summary

Herbicide-resistant weed populations are evolving rapidly worldwide and are the greatest challenge to current weed management strategies. By exerting intense selection pressure on weed populations, repeated overuse of certain herbicides has allowed for herbicide-resistant plants to survive and their densities to increase over time. Understanding the mechanisms of herbicide resistance development and spread gives farmers the tools to detect the early warning signs of resistance, take appropriate actions to control suspected resistant plants, and implement strategies to avoid or delay herbicide resistance.

Introduction

Repeatedly using herbicides that target the same plant physiological processes has led to the selection of plants that can naturally survive applications of these herbicides (Vencill et al. 2012).

Herbicides place tremendous selection pressure on weeds by killing susceptible individuals, but allowing naturally resistant individuals to survive and reproduce. The greatest number of herbicide-resistant weed species is reported for the acetolactate synthase (ALS) inhibitor, triazine, and acetyl CoA carboxylase (ACCase) inhibitor herbicides (Figure 8.1). Currently, 252 species of weeds present in 92 different crops and 69 countries have developed resistance to herbicides. Overall, weeds have evolved resistance to 23 of the 26 known herbicide sites of action, totaling 163 different herbicides (Heap 2018). Continual development and spread of resistant plants within some weed species poses a direct threat to the sustainability and the long-term survival of current cropping systems. The presence of herbicide-resistant weeds requires substantial changes in weed and crop management practices, increases the cost of weed control, and reduces the number of viable herbicide options. Understanding the origin and underlying causes of herbicide resistance gives farmers keys to avoid herbicide resistance development in weeds and maintains effective management tools.

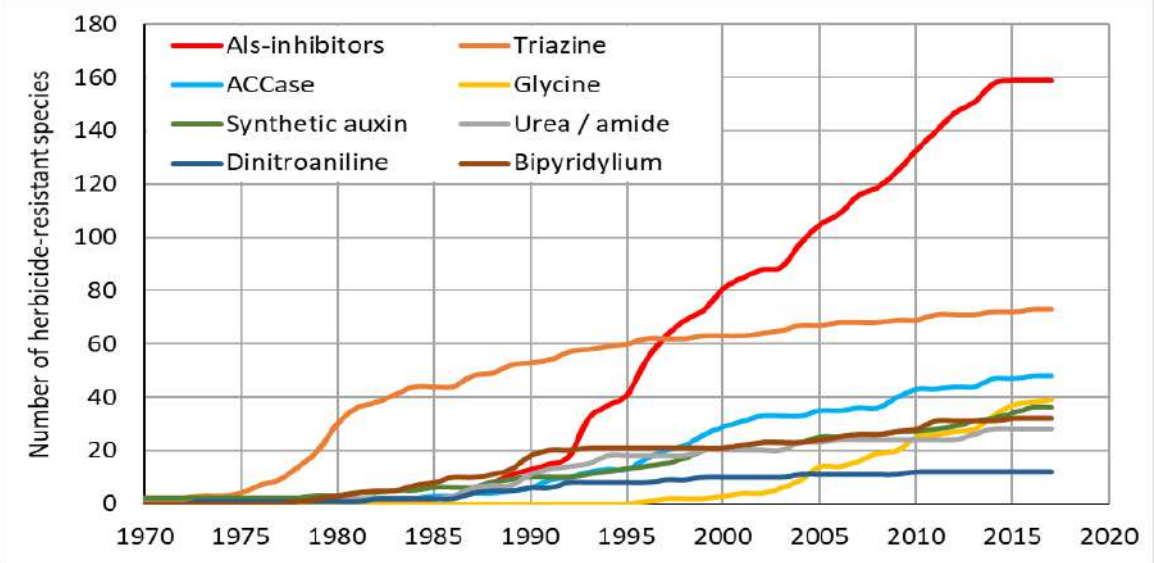

Figure 8.1. Chronological increase in resistant weeds for the most common herbicide families (Heap, 2017).

How Does Herbicide Resistance Develop?

Herbicide resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type (WSSA 1998). In simple terms, the herbicide no longer controls a weed population as it once did. While susceptible plants within a weed population will be killed, plants that naturally developed a genetic resistance to a specific herbicide will escape and reproduce. This is known as selection pressure: by killing all susceptible plants, the herbicide selects those that can survive an application of this herbicide. If this process continues for several weed generations, the populations of the herbicide-resistant weed will gradually increase until a noticeable portion of the population is no longer controlled by the herbicide. That is usually when farmers realize that herbicides once effective at controlling certain weeds no longer provide the expected level of control.

Mechanisms of Herbicide Resistance

Herbicide resistance mechanisms can be categorized as target-site or non-target site resistance. In target-site resistance, the molecular structure of the target site (the location within a plant where a herbicide acts to disrupt a plant process or function) is altered. The herbicide can no longer bind to its site of action (usually an enzyme) and interfere with plant physiological processes. This mechanism is considered the primary mechanism of resistance for herbicides that

are inhibiting the enzymatic activity of acetolactate synthase (ALS inhibitors), acetyl CoA carboxylase (ACCase inhibitors), and protoporphyrinogen oxidase (PPO inhibitors) (Powles and Preston 2006). This is also the mechanism involved with resistance to herbicides inhibiting cell mitosis (dinitroanilines) or photosynthesis (triazines) (Table 8.1).

Table 8.1. Number of target-site herbicide resistant weed species worldwide in 2018 for major herbicide sites of action (Adapted from Heap 2018).

Herbicide Site of Action	HRAC Group	Monocots	Dicots
ALS inhibitors	B	62	98
Photosystem II inhibitors	C1	23	51
ACCase inhibitors	A	48	0
EPSP synthase inhibitors	G	20	22
Cell mitosis inhibitors	K1	10	2
PPO inhibitors	E	3	10

Target-site resistance also can be caused by increased production of the targeted enzyme in resistant plants. According to research analyzing glyphosate-resistant Palmer amaranth, resistant plants produce more copies of the EPSP synthase enzyme targeted by glyphosate than susceptible plants. A higher number of enzyme copies means a labeled rate of glyphosate will not be sufficient for inhibiting enzymatic activity. By increasing its number of EPSP synthase copies, the plant survives a glyphosate rate that would otherwise be lethal to susceptible plants.

Non-target-site resistance is another mechanism through which plants can develop resistance to herbicides. As suggested by its name, this mechanism does not involve the herbicide active site. For example, the weed can increase its metabolic activity and eliminates the herbicide before it affects plant physiological processes. Weeds also can reduce the absorption of the herbicide active ingredient or limit the number of herbicide molecules that will actually reach the site of action by sequestering them within an inactive cellular site. The actions involved in non-target-site resistance may be expression of natural, enhanced tolerance to environmental stresses. Non-target-site resistance is often governed by many genes (polygenic) and may confer resistance to herbicides with different sites of action (Délye et al. 2013).

Selection Processes Leading to Herbicide Resistance

Although various environmental, biological, and human factors affect the timing of herbicide resistance onset and the speed of its spread, the way herbicides are used for controlling weeds is the most important factor leading to the evolution of herbicide resistance (Norsworthy et al. 2012). Repetitive use of a single herbicide or a group of herbicides with the same site of action favors the survival and development of plants naturally resistant to this site over those that are susceptible. Initially, a low number of individual plants with genetic adaptations giving them the ability to withstand a specific herbicide are present within a weed population. When this herbicide is applied,

susceptible plants are controlled, but resistant plants survive, grow, and produce seeds that contribute to the spread of herbicide resistance. This selection process continues with repeated applications of herbicides with the same site of action. The number of resistant individuals gradually increases, until the majority of the plants within a weed population are herbicide-resistant.

Reduced herbicide rates also can be a contributing factor to the evolution of herbicide-resistant weeds. Herbicide susceptibility varies among individuals within a weed population, allowing some plants to survive when exposed to a herbicide application. Reduced herbicide rates may allow plants with low or intermediate levels of resistance to survive. For instance, diclofop (Hoelon®) applied below the labeled rate was a major factor to the development of diclofop-resistant rigid ryegrass in Australia (Manalil et al. 2011). Genes that individually have a minor effect on the development of herbicide resistance can accumulate over time when herbicides are consistently sprayed at reduced rate. Cross-pollination recombines these genes, resulting in plants with higher levels of herbicide resistance than the previous generations (Délye 2013). Reduced rates may be the result of a weed management strategy or herbicide chemistry or formulation. For example, herbicide volatilization or slow degradation in the soil can expose weeds to sub-lethal herbicide rates. Reduced rates also can result from herbicide applied on plants larger or at a more advanced growth stage than recommended by the label. Crops with large canopy cover, inappropriate herbicide mixing, or inaccurate spray calibration result in insufficient spray coverage and reduced effective rates of weed control. Using the herbicide rate as indicated on the label and applying herbicides to weeds at the correct size are key to preventing herbicide resistance evolution and should be accompanied with proper weed scouting and sprayer calibration (see Chapter 4: *Weed Scouting and Mapping*).

Factors Affecting Resistance Development

Herbicide chemistry and its behavior in the soil or plant play an important role in the development of herbicide resistance. Herbicides that provide a high level of weed control eliminate a great portion of herbicide-susceptible weeds. Since only herbicide-resistant plants will survive and reproduce, resistance is more likely to develop in weeds that are highly susceptible to a specific herbicide because susceptible plants will be rapidly eliminated.

Herbicides that degrade slowly will place a greater selection pressure for resistance development because weeds are exposed to the herbicide for a longer period of time. Susceptible seedlings that emerge after the use of herbicide with no or short residual activity will survive, reproduce, and replenish the soil seedbank with herbicide-susceptible seeds. However, susceptible seedlings that emerge after the use of a long-residual herbicide will still be exposed to that herbicide, and only resistant biotypes will survive and reproduce.

Herbicides that target a single site of action will more likely favor the emergence and spread of herbicide-resistant weeds than those that interfere with multiple processes in the plant. For example, ALS-inhibitor herbicides (Group 2) specifically target the acetolactate synthase. Any structural change to this enzyme can confer resistance to the different herbicide families of ALS inhibitors. On the other hand, chloroacetamide herbicides (Group 15) interact with several enzymes involved in the biosynthesis of long-chain fatty acids. Targeting multiple sites of action may explain why resistance to chloroacetamide herbicides is relatively rare with only five known cases of resistant weeds. However, resistance to ALS inhibitors has been confirmed for 160 species worldwide.

What are Herbicide Groups?

Herbicide Resistance Action Committee (HRAC) and Weed Science Society of America (WSSA) have classified commercially available herbicides according to their sites of action, symptoms similarity, or chemical classes. HRAC uses letters and WSSA numbers to identify the various herbicide groups. For example, herbicides inhibiting photosynthesis at photosystem II are classified under group C by HRAC. Subclasses C1, C2 and C3 indicate different binding herbicide binding sites. These subclasses correspond to WSSA groups 5, 7, and 6, respectively.

Biology and genetics also are important factors in herbicide resistance development. The frequency of resistance in a weed population prior to herbicide application determines how long it takes for herbicide resistance to evolve. Resistance will spread faster with higher resistance frequencies. A 1:100,000 ratio of resistant weeds to total weeds will cause faster spread than a 1:10,000,000 ratio. Also, weeds with greater genetic diversity have greater chance of harboring resistance genes to a specific herbicide. For example, weeds belonging to the *Amaranthus* genus (or pigweeds) have considerable genetic diversity and some species have developed resistance to six herbicide sites of action (Heap 2018). Cross pollination and large seed production increase the risk of herbicide resistance dispersion. For example, Palmer amaranth male and female flowers are on separate plants, making cross pollination necessary for the production of seeds. Even plants that are 1,000 feet apart can transfer resistance to glyphosate from one to the other through pollen dissemination (Sosnoskie et al. 2012). Palmer amaranth averages 500,000 seeds produced per plant when there is no competition, allowing quick spread of glyphosate resistance.

Weed population size also contributes to the onset of herbicide resistance. The greater the number of plants exposed to a herbicide, the higher the risk of increased resistance genes frequency and resistance development (Gressel and Levy 2006). Preventing large weed populations and weed seedbank replenishment is a key component in herbicide-resistance management.

Types of Herbicide Resistance

Cross resistance occurs when a weed develops resistance to two or more herbicides that target the same site of action (Vencill et al. 2012). These herbicides can belong to the same or different herbicide families. For example, a single point mutation in the enzyme acetolactate synthase (ALS) of common ragweed may provide resistance to chlorimuron (Classic®) and cloransulam (FirstRate®). These herbicides belong to two different herbicide families, but have the same site of action (Figure 8.2).

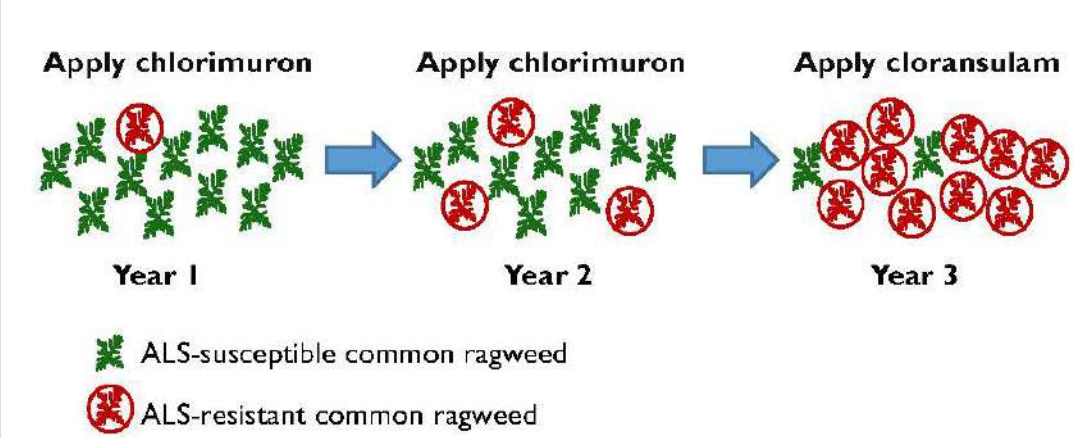

Figure 8.2. Example of cross-resistance in common ragweed, resistance to more than one herbicide with same mechanism of action. Chlorimuron is the active ingredient in Classic®; cloransulam is the active ingredient in FirstRate®. Both herbicides are ALS-inhibiting herbicides (or Group 2) (Weed Science Society of America, 2017).

Multiple resistance means that a weed is resistant to several herbicides with different sites of action (Powles and Preston 1995). For example, imagine that a farmer applies FirstRate®, an ALS-inhibitor herbicide (group 2), to control common ragweed (Figure 8.3). The repeated use of FirstRate® unintentionally selects for an ALS-resistant biotype (shown in black), which will dominate the common ragweed population and prevent effective ragweed control. The farmer then switches to Roundup®, a 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase inhibitor (group 9), and uses it continuously for several years. Continued Roundup® use selects for plants resistant to group 9 herbicides within

a population that is already resistant to group 2. Ultimately, the common ragweed population has developed multiple herbicide resistance with individuals that are resistant to both group 2 and group 9 herbicides.

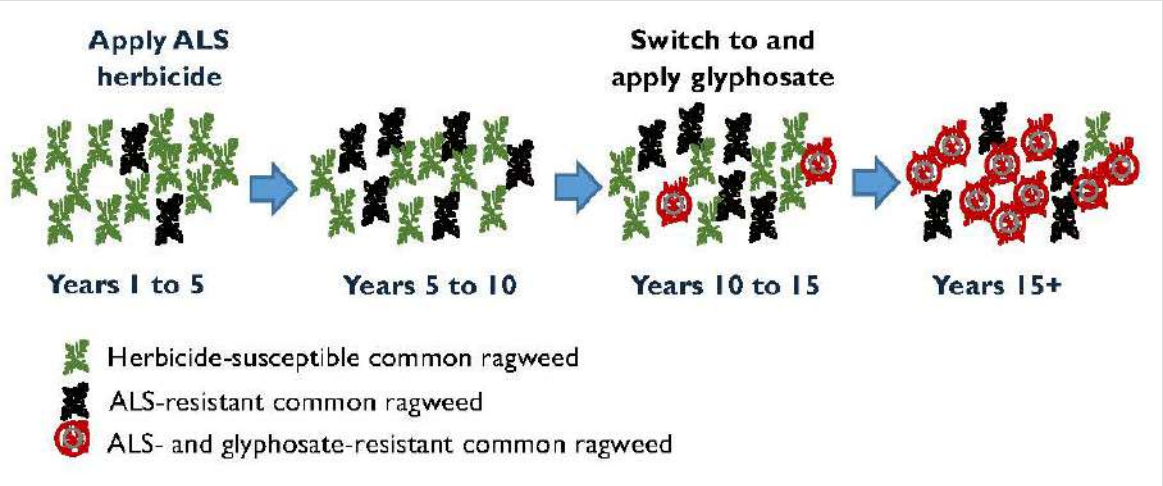

Figure 8.3. Example of multiple resistance in common ragweed, resistance to more than one herbicide mechanism of action. ALS-resistance are biotypes resistant to acetolactate synthase-inhibiting herbicides (or Group 2, i.e. Classic® or FirstRate®). Glyphosate is the active ingredient in Roundup® and is a Group 9 herbicide (Weed Science Society of America, 2017).

Weed Species Shifts and Weed Resistance

A weed species shift is a change over time in the relative abundance of the weed species that form a weed communities. These species are not equally affected when a specific herbicide is applied. Some species can be completely controlled while others are only partially controlled or not affected at all. Recurrent use of the same herbicide causes shifts toward species that are not vulnerable to this herbicide. For example, the continued use of broadleaf herbicide 2,4-D in cereal grain crops eventually leads to the elimination of susceptible broadleaf weeds. Grassy weeds that are tolerant to 2,4-D will survive, multiply, and dominate the weed communities over time. In this scenario, 2,4-D selectivity is caused by differential physiological sensitivity between grassy and broadleaf weeds, not because of resistance to 2,4-D (Figure 8.4). Weed species shifts are not only driven by herbicide use; they also may be the result of other agronomic practices such as tillage, crop rotation, or nonchemical weed control tactics.

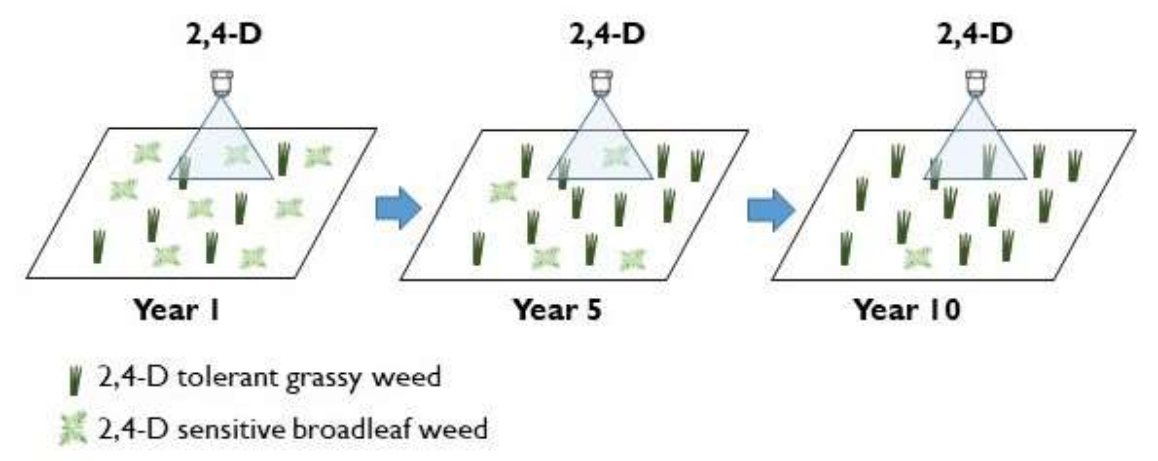

Figure 8.4: Example of a weed species shift due to 2,4-D application and grass tolerance to 2,4-D (Weed Science Society of America, 2017).

Spread of Herbicide Resistance

Once established, herbicide-resistant weeds can easily spread through the dispersal of pollen, seeds or plant parts that can easily re-root.

The movement of pollen from herbicide-resistant plants to susceptible plants as cross-pollination results in seeds that may carry the resistance gene(s). If these seeds ripen and replenish the soil seedbank, the resulting plants could survive herbicide application and further disseminate resistance through seed production. The risk of resistance spreading is higher for cross-pollinating weeds, such as pigweed, than for self-pollinating species, such as grasses. The distance that pollen can travel is determined by pollen grain size, wind velocity, the weeds ability to attract pollinators, the amount of pollen available for dispersal, and the length of time that pollen is viable for pollination. The only way farmers can prevent the spread of herbicide resistance through pollen is to eliminate suspected resistant weed species from fields and surrounding areas prior to weed bloom time.

Spread of seeds from herbicide-resistant populations is similar to herbicide-susceptible populations. See Chapter 2: *Identification and Characteristics of Weeds* and Chapter 6: *Prevention of Weeds* for information on weed seed spread and how to prevent it.

Resistance Avoidance Strategies

Herbicide resistance will evolve in a weed population if two conditions are satisfied:

1. Plants with a naturally occurring mutation that confers resistance to a specific herbicide group are already present in the weed population, and
2. This herbicide group is used extensively on these plants without use of another effective weed control tactic.

Resistance mutations within the plants are impossible to see until inadequate weed control is noticed because resistance traits are not visible on the plant. Preventing resistance issues requires reducing opportunities for resistant individuals to survive and reproduce.

Applying and rotating herbicides wisely will prevent the selection of herbicide-resistant plants. The best method is to rotate effective herbicide sites of action, either by applying multiple effective sites of action within a given crop or by alternating crops with different labeled herbicides, such as corn and soybean, between cropping seasons. Combining sites of action, either by mixing effective herbicides or applying them sequentially, can control weeds resistant to a given site of action, or delay the onset of herbicide resistance. Using labeled herbicide rates prevents the proliferation of plants that would survive at a sub-lethal rate. Also, following label recommendations about the size of the weeds when applying a postemergence herbicide is critical. Spraying plants taller than the maximum recommended size reduces the amount of herbicide that reaches the plant and results in a sub-lethal rate being applied. Non-uniform spray distribution, which can be caused by improper sprayer calibration or excessive weed density, may also decrease herbicide effectiveness and select plants that can survive a sub-lethal dose.

Herbicide-resistant management requires using two **effective** sites of action, applied at the full rate, and applied at the right timing. Tank mixing effective sites of action is more beneficial than applying the herbicides in sequence.

The rapid detection of weed resistance is crucial and relies on efficient weed scouting techniques (see Chapter 4: *Weed Scouting and Mapping*). Signs that a weed population in a field may be herbicide-resistant include the following:

- Poor herbicide performance on one weed species but not others, even though the herbicide is known to control this species.
- Patchy distribution of weeds that survived the application of herbicide that otherwise should have controlled them.
- A majority of individuals within a weed species have been controlled with an efficient herbicide while others have escaped control.

All possible actions to prevent herbicide resistant suspected plants from producing seeds should be considered. Options include hand weeding, use of an

efficient herbicide, or mechanical elimination of all surviving weeds within the affected areas which also may include crop destruction.

Key Points

- Overuse of a single herbicide mode of action may lead to the proliferation of individual weeds that can survive its labelled rate which otherwise is lethal on susceptible plants.
- Resistance can be caused by structural modification of the herbicide target within the plant (target-site) or by other metabolic or exclusion mechanisms (non-target-site).
- Resistance can result in the dominance of one weed species and the exclusion of other species.
- Environmental factors and human-related activities can contribute to herbicide-resistant weeds spreading over large distances.
- Wise use of herbicides, weed management diversification, and early detection of resistant weeds are key strategies in preventing the development and spread of herbicide resistance.

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