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Herbicide Resistance and Mechanism of Resistance in Weeds

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Globally, the rapid evolution of herbicide resistance has become a major challenge to sustainable crop production. Herbicide resistance may occur as cross-and multiple resistance. In case of cross-resistance a single resistance mechanism confers resistance to two or more herbicides same mechanism of action. While, in multiple resistance, a weed population possesses two or more distinct resistance mechanisms, enabling it to survive herbicides with different modes of action. The first documented case of herbicide resistance was reported in *Senecio vulgaris* L. to the a triazine herbicide simazine, a PSII inhibitor. According to the latest records, 548 unique cases of herbicide-resistant weeds (species × herbicide site of action) have been confirmed globally, involving 275 weed species, including 156 dicotyledonous and 119 monocotyledonous species (Heap, 2026). Resistance has evolved to 21 of the 31 known herbicide sites of action, and have been reported in 102 crops across 76 countries, highlighting the widespread occurrence and increasing global importance of herbicide resistance.

Mechanism of herbicide resistance

There are several mechanisms like reduction in the target site sensitivity and herbicide activation; overexpression or amplification of target-site gene; enhanced herbicide detoxification; restricted absorption or translocation, and sequestration of the herbicide away from its target site. The mechanism works in isolation or integrated form results in development of herbicide resistance in weed species. Understanding the underlying basis of herbicide resistance is essential for developing effective management strategies. Herbicide resistance is mediated through two major mechanisms: target-site resistance (TSR) and non-target-site resistance (NTSR).

Target-site resistance (TSR)

TSR is caused by genetic alterations in the herbicide target gene that reduce the sensitivity of the target protein to herbicide inhibition while maintaining adequate enzymatic function. The principal mechanisms include amino acid substitutions, codon deletions, target-gene amplification, and target-gene overexpression, which decrease herbicide binding affinity or increase the amount of target enzyme available for inhibition. The level of resistance conferred depends on the specific mutation and its effect on herbicide binding affinity and enzyme function, ranging from moderate to high levels of resistance Target-site resistance is generally site-of-action specific, conferring resistance only to herbicides that inhibit the same target enzyme. In addition to target-site mutations, target-gene amplification and overexpression represent important mechanisms of target-site resistance. Gene duplication, defined as the heritable replication of a DNA segment resulting in additional gene copies, is a major driver of this mechanism. Consequently, the abundance of the target enzyme exceeds the inhibitory capacity of field-recommended glyphosate doses, thereby conferring resistance.

Nontarget-site mechanisms (NTSR)

Non-target-site resistance comprises all resistance mechanisms that limit the amount of biologically active herbicide reaching the site of action or mitigate herbicide toxicity without modification of the target protein. These mechanisms include reduced absorption, impaired

translocation, vacuolar sequestration, enhanced metabolic detoxification, and, less commonly, reduced herbicide activation. Because NTSR mechanisms may confer resistance to herbicides with different sites of action, they often result in cross-resistance or multiple resistance, posing a major challenge for sustainable weed management.

Foliar absorption is largely influenced by leaf surface characteristics, including cuticle thickness, cuticle composition, epicuticular waxes, trichomes, and leaf morphology, which determine herbicide retention and penetration. While reduced foliar absorption alone generally confers only low levels of resistance, it can substantially enhance resistance when combined with other target-site or non-target-site mechanisms. In tolerant grass species, rapid metabolic detoxification of 2,4-D through hydroxylation and subsequent conjugation, together with reduced sensitivity of auxin response pathways, limits herbicide activity and confers natural tolerance.

Reduced translocation is an important non-target-site resistance mechanism in which herbicide movement from source leaves to sink tissues is restricted, thereby reducing the amount of biologically active herbicide reaching the site of action. This may result from vacuolar sequestration, accumulation in leaf trichomes, or altered activity of plasma membrane transporters, particularly ATP-binding cassette transporters, which sequester herbicides into vacuoles. Reduced glyphosate translocation was first demonstrated in glyphosate-resistant *L. rigidum* from Australia. This is due to differences in cellular distribution of glyphosate and subsequent phloem loading and translocation. The *phoenix phenomenon* is an unusual mechanism of reduced herbicide translocation in which rapid herbicide-induced tissue injury restricts movement of the herbicide to the meristems.

The selectivity of many herbicides relies on the greater capacity of crop plants to detoxify herbicides into non-toxic metabolites more rapidly than susceptible weeds. This inherent difference in metabolic capacity forms the basis of selective chemical weed control and also underlies the evolution of metabolism-based non-target-site resistance in weed populations. Enhanced metabolism is divided into four phases, viz., (i) conversion, (ii) conjugation, (iii) transportation, and (iv) deposition. In many cases, weeds possess the same herbicide detoxification pathways as related crop species, but these genes are typically expressed at lower levels.

Herbicides such as ALS and ACCase inhibitors are selectively toxic because crops detoxify them more rapidly than susceptible weeds. This detoxification is primarily mediated by cytochrome P450 monooxygenases (P450s), glutathione S-transferases, and glycosyltransferases. Among these, glutathione-S-transferases are detoxification enzymes that conjugate herbicides with glutathione, facilitating detoxification and sequestration. For example, in wheat, the ACCase inhibitor diclofop-methyl is detoxified through P450-mediated hydroxylation followed by GT-mediated glucose conjugation, whereas susceptible weed species metabolize the herbicide too slowly to prevent phytotoxicity

Management of herbicide resistant weeds

- Harvest weed seed control (HWSC), effectively destroys weed seeds during crop harvest, preventing their return to the soil seed bank. Harvest weed seed control (HWSC) that removes weed seeds along with crop residues, concentrating weed seeds into narrow windrows or chaff lines, or destroying them during harvest using impact mills prevents deposition of weeds seed in soil seed bank. The system has been reported to destroy 93–99% of seeds of wild radish, wild oat, brome grass, and annual ryegrass (Walsh et al., 2013). By reducing annual seed rain and soil seed bank replenishment, HWSC is an effective tool for integrated weed management and delaying herbicide resistance evolution.
- Preventive, cultural, and mechanical practices are integral components of integrated weed management. These include the use of weed-free seed, farm machinery sanitation, prevention of weed seed spread, and control of weeds on field margins, bunds, and irrigation channels. Cultural practices such as the stale seedbed technique, zero tillage with residue retention, competitive crop cultivars, higher seed rates (up to 125 kg ha^{-1}), and

narrow row spacing (18–20 cm) in wheat improve crop competitiveness, suppress weed establishment, reduce soil seed bank replenishment, and delay the evolution of herbicide resistance. Crop rotation diversifies weed communities and reduces herbicide selection pressure by altering tillage practices, herbicide programs, and crop management (crop establishment, seed rate, fertilization, and irrigation). These changes disrupt weed life cycles, suppress weed infestation, and delay herbicide resistance evolution.

- The identified alternative herbicides can reduce the selection pressure for cross- and multiple-herbicide resistance in weeds. However, sustainable resistance management requires an integrated weed management approach that includes rotation of herbicides with different modes of action, crop and machinery sanitation, weed seed harvest control to minimize soil seed bank replenishment, and proper herbicide application practices (recommended dose, appropriate nozzle selection, and timely application). In addition, farmer surveys, field demonstrations, and extension programs are essential to assess field-level resistance and promote awareness of effective herbicide stewardship.
- Emerging biotechnological tools, such as RNA interference (RNAi) and CRISPR-mediated gene editing, offer promising alternatives for the management of herbicide-resistant weeds. RNAi functions by silencing genes responsible for herbicide resistance, while CRISPR facilitates targeted genome editing to improve weed sensitivity to herbicides. Although no single weed management program can provide complete control under all conditions, the adoption of diverse, site-specific management practices can substantially reduce weed proliferation and delay the evolution of herbicide resistance.
- Integrating multiple weed control methods is essential for reducing reliance on herbicides and minimizing the chemical footprint of weed management.

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