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Herbicide resistance in weeds: a serious matter to concern.

Author: SIRAZUDDIN

Herbicide resistance is the inherited ability of a biotype of a weed to survive an herbicide application to which the original population was susceptible. In simple terms, resistance refers to a situation where a given herbicide, applied at the recommended rate and time, once controlled a particular weed population but, after repeated use, that herbicide no longer controls that population. That population is said to be resistant (or resistance has developed in that population).

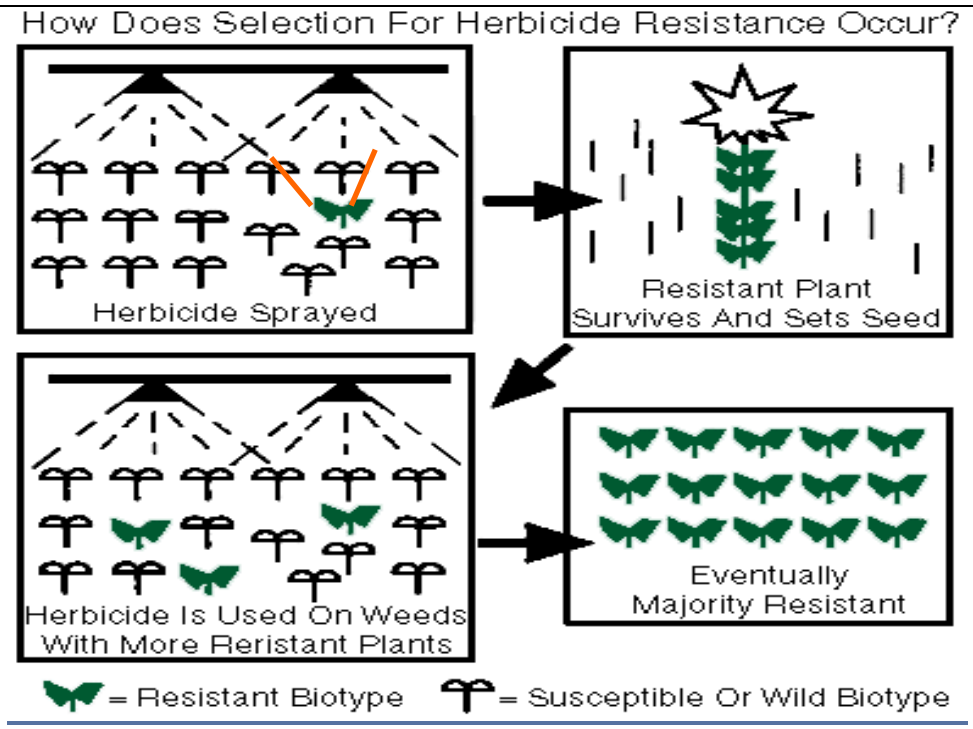
Types of herbicidal resistance

- **Cross resistance**: Weed biotype that has gained resistance to more than 1 herbicide with the same mode of action but same or different families.

- **Multiple resistance**: Weed biotype that has developed tolerance to more than one herbicide brought about by different selection pressures *(different modes of action).*

Conditions favoring herbicide resistance

- Repeated use of a specific herbicide or a combination of herbicides
- Weed populations with wide genetic diversity may develop resistance rapidly, especially for herbicides with a single mechanism of action
- Weed possessing characters like large plant numbers, prolific seed production, high rates of weed migration/spread, and diverse environmental conditions may contribute to high genetic diversity and develop resistance very quickly

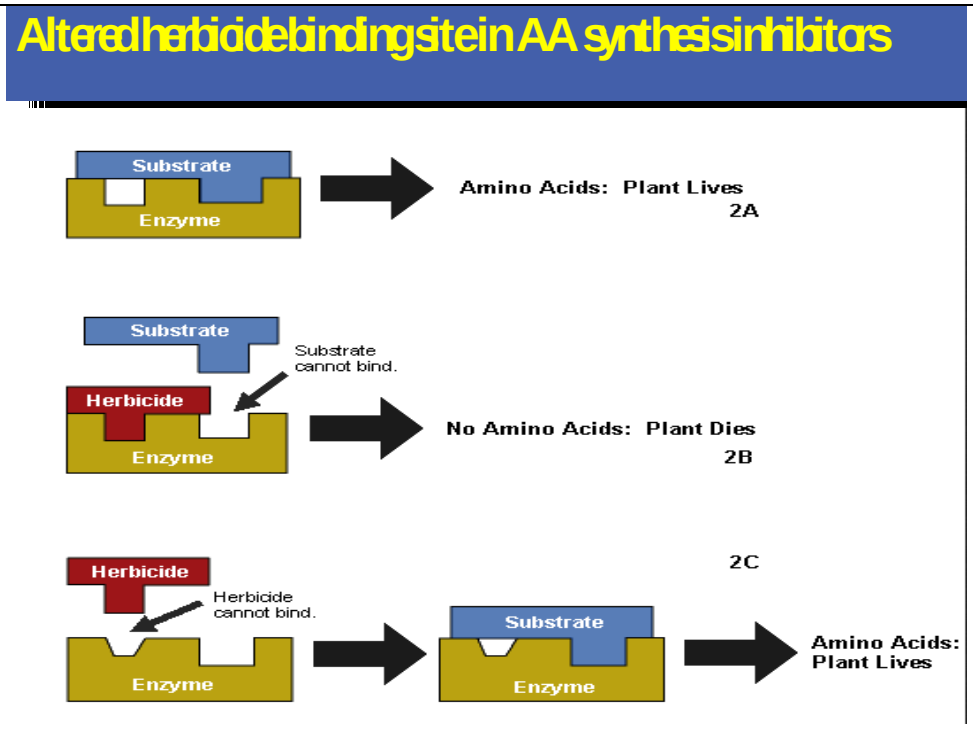


The four known mechanisms of resistance to herbicides are:

1. Altered target site
2.Enhanced metabolism
3. Compartmentalization or sequestration
4. Over-expression of the target protein

Altered target site

- An herbicide has a specific site (target site of action) where it acts to disrupt a particular plant process or function (mode of action). If this target site is somewhat altered, the herbicide no longer binds to the site of action and is unable to exert its phytotoxic effect. This is the most common mechanism of herbicide resistance.
- Where the herbicide has such little inhibitory effect on the site of action, plants may survive greater than 10 times the normal herbicide rate (considered high-level resistance).
- Mechanisms of action where high-level resistance is most often seen include ACCase, ALS, and photosystem II inhibitors.



Enhanced metabolism

- This type of resistance is **more complex** than altered site-of-action type resistance because it involves several plant processes.
- Plants with altered metabolism resistance can **degrade several unrelated herbicides of different modes of action** through multiple genes controlling metabolic processes.
- Plant injury may occur because plants cannot rapidly degrade absorbed herbicide, causing this mechanism to be considered low-level resistance.
- Increasing the herbicide rate to smaller plants may control more plants.

Examples:

- ✓ Ryegrass resistant to Acetyl coenzyme A carboxylase, Acetolactate synthase , and photosystem II inhibitors.
- ✓ Velvetleaf resistant to atrazine.
- ✓ In simazine resistance, the herbicide is acted upon by cytochrome P-450 monoxygenase enzyme and converted to herbicidally inactive de-ethyl simazine and di-de-ethyl simazine
- ✓ Simazine resistance in *Lolium rigidum*

Compartmentalization or sequestration

- Some plants are capable of restricting the movement of compounds (herbicides) within their cells or tissues to prevent the compounds from causing harmful effects. In this case, an herbicide be inactivated either through binding (such as to a plant sugar molecule) or removed from metabolically active regions of the cell to inactive regions, eg. cell wall, where it exerts no effect.

- Nearly all plants with this type of resistance are injured shortly after the herbicide application because the herbicide cannot be moved away from the site of action fast enough and for a long enough time.
- Herbicide sequestration is considered low-level resistance because increasing rates applied to smaller plants increases mortality.

Examples: Glyphosate-resistant biotypes of horseweed, ryegrass, common and giant ragweed.

Over-expression of the target protein

- If the target protein, on which the herbicide acts, can be produced in large quantities by the plant, then herbicide becomes insignificant.

Most Important Herbicide-Resistant Species

1.	Rigid Ryegrass	*Lolium rigidum*
2.	Wild Oat	*Avena fatua*
3.	Redroot Pigweed	*Amaranthus retroflexus*
4.	Common Lambsquarters	*Chenopodium album*
5.	Green Foxtail	*Setaria viridis*
6.	Barnyardgrass	*Echinochloa crus-galli*
7.	Goosegrass	*Eleusine indica*
8.	Kochia	*Kochia scoparia*
9.	Horseweed	*Conyza canadensis*
10.	Smooth Pigweed	*Amaranthus hybridus*

Ten important herbicide mode of actions as per WSSA

Mode of action	WSSA group	No of resistant species
Acetyl coenzyme A carboxylase	Group 1	44
Acetolactate synthase	Group 2	142
Shoot inhibitors	Group 3	
PGR	Group 4	12
PS-II	Group 5	31
PSP	Group 9	72
PS-I	Group 22	25
Glutamine Synthetase	Group 10	29
Protoporphyrinogen Oxidase	Group 14	2
Cellulose inhibitors	Group 20/29	6

How to Prevent or Delay Herbicide Resistance

- Herbicide rotation
- Crop rotation
- Monitoring after herbicide application
- Non-chemical control techniques
- Short-residual herbicides
- Certified seed
- Clean equipment

Proactive Herbicide Resistance Management

- Early detection of resistance means management will be easier, and it increases the potential to avoid the spread of the resistant biotype. Unfortunately, because resistant plants and susceptible plants look alike, resistance often is not detected until the resistant biotype has spread to 30% or more of the field and perhaps to surrounding fields.
- Therefore, a proactive approach using diverse weed control tactics is the most effective way to manage herbicide resistance.
- The primary objective of proactive resistance management is to reduce selection pressure by:

1) selecting and using herbicides correctly;
2) recognizing weed characteristics that promote resistance; and,
3) Managing fields, farms, or sites wisely.

References (if any)

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