HERBICIDE RESISTANCE IN WEEDS AND ITS MANAGEMENT

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Article is about Herbicidal resistance and its types and mechanisms of herbicidal resistance and ways and means to control the resistance

Article

HERBICIDE RESISTANCE

Altered response of a formerly susceptible weed species to the extend that some individuals in the species are no longer susceptible.

HISTORY OF HERBICIDE RESISTANCE

- The resistance in weed *Senecio vulgaris* L. (Common groundsel) to triazines was detected in USA in 1968 and was reported by Ryan in 1970.
- In India, *Phalaris minor* developed resistance to isoproturon during 1992-93, and the same was reported by Malik and Singh (1995).This was the most serious case of herbicide resistance in the world.

TYPES OF HERBICIDE RESISTANCE

1) Cross-resistance
2) Negative cross resistance/ collateral
3) Multiple resistance
4) Reverse resistance
5) Co-resistance/compound resistance

1) Cross-resistance

When resistance in weed is evolved due to continuous use of same or more herbicides having similar mode of action/single resistance mechanism.

Common cocklebur resistant to one herbicide (eg. Classic, chlorimuron) may also be resistant to another herbicide (e.g. Scepter, imazaquin) with the same mode of action (ALS-inhibitor). This is known as cross-resistance. Photo courtesy of Dallas Peterson.

2) Negative cross resistance/collateral

Mechanism by which an individual resistant to one herbicide or a chemical family of herbicides shows higher or increased sensitivity to other herbicides than its natural wild type susceptible population.

Example:

Resistant biotype of *Phalaris minor* controlled more effectively by fenoxaprop-p-ethyl than susceptible wild type.

3) Multiple resistance

When resistance to several herbicides results from two or more distinct resistance mechanism in the same plant.

Common waterhemp {A*maranthustuberculatus (syn. rudis)*} seedlings were identified as having multiple resistance, that is, resistance to both triazine and ALS-inhibiting herbicides, which have two different modes of action with different mechanisms conferring resistance. Photo courtesy of Dallas Peterson.

4) Reverse resistance

Phenomenon in which the weed biotypes resistant to a herbicide falls susceptible to the every herbicide if it is not used for a period of 7 - 10 years, instead some alternative herbicides are used to kill the resistant population.

5) Co-resistance/compound resistance

When weed develops resistance to both mixing partner herbicides of a mixture applied concurrently.

Loliumrigidum developed resistance to Amitrol and Atrazine applied concurrently.

STATUS AND DISTRIBUTION OF HERBICIDE RESISTANT WEED BIOTYPES

Sr.	Mode of action	Herbicide	Biotypes
1	Inhibition of ALS/AHAS	Chlorsufuron	95
2	Inhibition of photosynthesis at PS II	Atrazine	67
3	Inhibition of ACCase	Diclofop methyl	35
4	Synthetic of auxins (Making IAA)	2,4-D	26
5	PS I electron diversion	Paraquat	24
6	Inhibition of photosynthesis at PS II	Chlorturon	21
7	Inhibition of EPSP synthase	Glyphosate	14
8	Microtubule assembly inhibition	Trifluralin	10
9	Inhibition of lipid synthesis (not ACCase)	Triallate	8
10	Bleaching : inhibition of carotenoid synthase	Amitrol	4
11	Inhibition of protoporphyrinogen oxidase	Oxyflurfen	3
12	Unknown	Flamprop methyl	2
13	Bleaching: Inhibition of carotenoid biosynthesis at phytoenedesaturase step (PDS)	Flurtamore	2
14	Inhibition of cell division	Butachlor	3
15	Inhibition of photosynthesis at PS II	Bromoxynil	1
16	Inhibition of mitosis or microtubule polymerization	Propham	1
17	Inhibition of cell wall (cellulose) synthesis	Dichlobenil	1
18	Unknown	Difenzoquat	1

19	Unknown	MSMA	1
Total			**319**

COUNTRY WISE HERBICIDE-RESISTANT WEED BIOTYPES EXISTING IN THE WORLD TILL JULY, 2008

Country	Weed biotypes
U.S.A.	122
Autralia	51
Canada	44
France	32
Spain	30
U.K.	24
Israel	23
Germany	19
Japan	16
South Africa	14
China	9
Iran	5
India	3

RESISTANT WEED SPECIES TO VARIOUS HERBICIDES

Sr. No.	Herbicides	Weeds
1.	Triazine	Amaranthus spp., Chenopodium album, Echinochloacrusgalli, Poaannua
2.	Dalapan	Cynodon dactylon, Echinochloacrusgalli, Sorghum halepense
3.	2,4-D	Cirsiumarvense, Daucuscarota
4.	Diclofop-methyl	Alopecurusmyoscuroids, Avenafatua, Loliumrigidium
5.	Paraquat and diquat	Loliumperene, Poaannua
6.	Propanil	Echinochloacrusgalli, Echinochloacolonum
7.	Chlorsulfuron	Loliumrigidium, Kochiascoparia
8.	Trifluralin	Eleusine indica, Setariaviridis
9.	Isoproturon	Phalaris minor
10.	Glyphosate	Cirsiumarvense

CAUSES FOR DEVELOPMENT OF HERBICIDE RESISTANCE

1. Over reliance on herbicides for weed control due to unavailability of labour and more labour wages

Pesticide	India %		World %	
	1997	2008	1997	2008
Herbicide	16	20	47	52
Insecticide	52	61	29	7

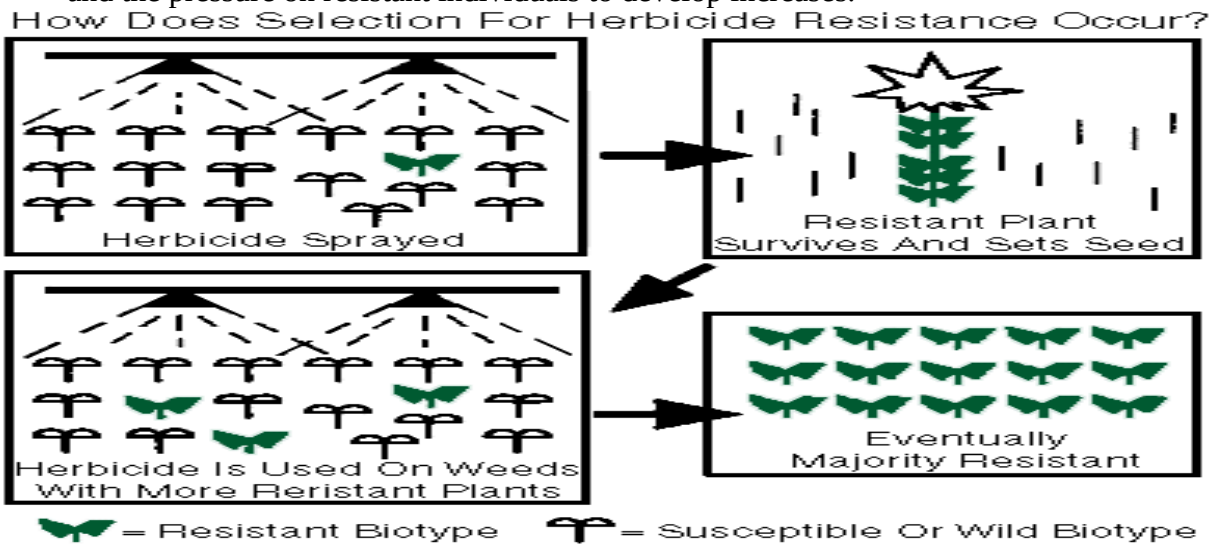
Fungicide	30	17	19	32
Others	2	2	5	4

2. Repeated use of same herbicide or other herbicides having same mechanism of action
3. Crop monoculture
4. Minimum/zero tillage

FACTORS OF HERBICIDE RESISTANCE

a) **Initial frequency:** Resistant genotypes are present in natural plant population in varying frequency

b) **Selection pressure:** When the herbicides are applied, the susceptible individuals are killed and the pressure on resistant individuals to develop increases.



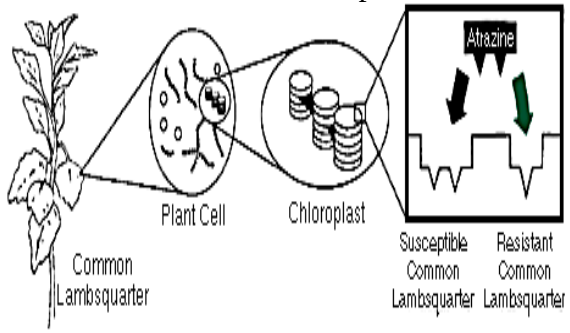
c) **Fitness:** Measures the potential evolutionary success of a genotype.

d) **Seed bank in soil:** Strong buffering influence in delaying the rate of appearance of resistance.

MECHANISM OF HERBICIDE RESISTANCE

1) An alteration of the herbicide site of action :

- In resistant biotypes the herbicide binding site of action is modified due to genetic change and this mechanism is responsible for resistance in most of the triazines, ALS inhibitors and dinitroaniline herbicides.
- Triazine herbicides are primarily photosynthetic inhibitors and in resistant biotypes, loss of herbicide binding ability occurs due to alteration of the binding site on thylakoid membrane of the chloroplast from serine to glycine at amino acid position 264.



The photosynthetic process occurs within a plant cell's chloroplasts. Certain herbicides can inhibit photosynthesis by binding to specific sites within the chloroplast. The relationship of a herbicide to the chloroplast binding site is very specific and any modification of the herbicide or binding site can eliminate herbicidal activity.

2) Enhanced metabolism:

Rapid degradation and or conjugation of herbicides into non-toxic or less toxic form are major mechanism of resistance in several weed species due to activation of certain enzymes

Example:

- Resistance to atrazine in some population of *Abutiliontheophrasti*is due to increased activity of glutathione-s-transferase that detoxifies atrazine.
- Resistance to propanil in *Echinochloacolona*is due to increased activity of enzyme arylacylamidase that detoxifies propanil.
- Isoproturon resistant in *Phalaris minor* is due to rapid degradation catalysed by cytochrome P-450 monoxygenase.

3) Differential translocation:

The less uptake of herbicide by the resistant biotypes generally results in less translocation of herbicides.

In *Loliumrigidium*, the resistant biotypes shows less translocation of chlorsulfuron compared with susceptible ones.

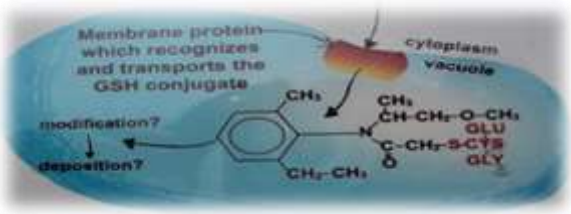
4) Sequestration and compartmentation:

Storing, accumulation or sequestration of the herbicides or their toxic metabolites in cell vacuoles, cells or tissues far from site of action and thus prevent them from reaching to site of action. (*Glutathione sulphyhydryl*)

Example:

Some lipophilic herbicides may become immobilized by partitioning into lipid rich glands or oil bodies (Stegink and Vaughn, 1988).

One of the major mechanism of resistance to paraquat is compartmentation in *Poaannua.*



MANAGEMENT OF HERBICIDE RESISTANCE

As resistance is essentially irreversible, therefore, it is virtually impossible to reintroduce susceptibility into that community to its pre-resistance level. Hence, it is important to start managing herbicide resistant weeds in initial stage of detection otherwise it may have serious consequences.

Main principle is selection pressure for evolution of resistance, therefore, main focus should be on modifying those factors and practices which are responsible for quicker evolution of resistance.

- Over reliance on herbicides for control of weeds
- Repeated use of same herbicide or other herbicides having same mechanism of action
- Crop monoculture
- Minimum/zero tillage

1) Use of alternative herbicides:

Immediately after detecting the herbicide resistance the use of that particular herbicide should be stopped.

Isoproturon – resistant *P. minor* has been effectively controlled by application of pendimethalin, clodinofop, fenoxaprop and sulfosulfuron.

2) Herbicides mixture and rotation:

The use of two or more herbicides having different mechanisms of action when used in mixture or rotation reduces the selection pressure or resistant biotypes and delay the rate of evolution as compared to individual herbicide used alone.

RECOMMENDED HERBICIDE MIXTURES FOR DIFFERENT CROPS

Crop	Herbicide mixture
Wheat	Diclofop-methyl 750 g + Fluroxypyr 100 g/ha
	Diclofop - methyl 750 g + Isoproturon 500 g/ha
	Isoproturon 750 g + tribennuron 100 g/ha
	Isoproturon 750 g + 2,4- D 500 g/ha
	Tralkoxydim 350 g + 2,4 –D 500 g/ha
Rice	Anilofos 0.4 kg + 2,4 –DEE 0.50 kg/ha
	Butachlor 1.0 kg + Propanil 2.0 kg/ha
	Butachlor 1.0 kg + 2,4-DEE 0.5 kg/ha
	Benthiocarb 1.0 kg + Chlorimuron 4 g/ha
	Anilofos 300 g + Metsulfuron-methyl 4 g/ha

3) Herbicide selection and application:

The record of herbicide resistant weeds reveals that few weeds have evolved resistance to chloracetamides, diphenyl ether and glyphosate despite extensive use of these herbicides. Therefore, they are considered as a low risk for the evolution of herbicide resistance in weeds.

Isoproturon – resistant P. minor has been effectively controlled by application of pendimethalin, clodinofop, fenoxaprop and sulfosulfuron.

Herbicide group	Degree of risk
Group A to B	Has high risk for development of resistance
Group C to H	Moderate risk for development of resistance
Group I to H	Low risk for development of resistance

CLASSIFICATION OF HERBICIDE BASED ON MODE OF ACTION

Group	Mode of action	Chemical family	Active ingredient
A	Inhibition of acetyle CoA carboxylase (ACCase)	Aryloxyphenoxy propionates	Clodinofop-propargyl
		Cyclohexannediones	sethoxidim
B	Inhibition of acetolactate synthase (ALS/AHAS)	Sulfonylureas	Chlorimuron-ethyl
		Imidazolinones	Imazathathyr
		Triazolopyrimidines	Diclosulam
		Pyrimidinylthiobenzoiates	Pyriminobac-methyl
C1	Inhibition of photosynthesis at photosystem II	Triazines	Atrazine, simazine
		Triazinones	Metribuzin
		Pyridazinone	Pyrazon
		Phenyl carbamates	Desmediphem
C2	Inhibition of photosynthesis at photosystem II	Ureas	Isoproturon, Diuron
		Amide	Propanil

C3	Inhibition of photosynthesis at photosytem II	Nitriles	Bromoxynil
		Benzothiadiazole	Bentazon
D	Photosystem I electron diversion	Bipyridiliums	Diquat, paraquat
E	Inhibition of Protoporphyrinogen oxidase	Diphenyl ether	Lactofen, oxyflourfen
		Thiadiaazoles	Thidiazim
		Oxadiazole	Oxadiazon
		Triazolinone	Cafentrazone
		Pyridazinone	Norflurazone
F1	Bleaching inhibition of caretenoidbiosynthesis	Nicotinanilie	Diflufenican
		Others	Flurtamone
F2	Bleaching inhibition of 4-hydroxyphenyl-pyruvate-dioxygenase (4-HPPD)	Triketone	Sulcotrione
		Isoxasole	Isoxaflutole
F3	Bleaching inhibition of caretenoid biosynthesis (unknown target)	Pyrazole	Pyrazoynate
		Triazole	Amitrole
		Isoxazolidinone	Clomazone
G	Inhibition of EPSPS synthase	Glycines	Glyphosate, sulfosate
H	Inhibition of glutamine synthase	Phosphinic acids	Glufisinate-ammonium
I	Inhibition of DHP (dihydropteroate) synthase	Carbamate	Asulam
K1	Microtubule assembly inhibition	Dinitroanilines	Trifluralin, pendimethalin
		Phosphoroamidate	Amiprophos-methyl butamiphos
		Pyradazines	Dithiopyr
		Benzoic acid	DCPA
K2	Inhibition of mitosis	Carbamates	Propham
		Benzyl ether	Cinmethalin
K3	Inhibition of cell division	Chloro-acetamides	Alachlor, Butachlor
		Carbamate	Carbetamide
		Acetamides	Diphenamid
		Benzamides	Propyzamide
		Oxyacetamids	Fluthiamid
L	Inhibition of cell wall (cellulose) synthesis	Nitriles	Dichlobenil
		Benzamide	Isoxaben
M	Uncoupling (membrane disruption)	Dinitrophenols	DNOC, dinoseb
N	Inhibition of lipid	Thiocarbamates	EPTC,

	synthesis not ACCase inhibition	Phosphorodithioate	Bensulide
		Benzafuran	Ethofumesate
		Cholo carbonic acid	TCA, dalapon
O	Synthetic auxins (IAA)	Phenoxy-carboxylic acids	2,4-D, 2,4,5 – T, MCPB
		Benzoic acid	Dicamba
		Pyridie carboxylic acids	Clopyralid
		Quiniline carboxylic acids	Quinclorac
		Others	Benazolin-ethyl
P	Inhibition of indoleacetic acid action	Phthalamate	Naptalam
		Semi-carbazone	Diflufenzopyr
Z	Unknown	Arylamio-propionic acid	Flaprop-methyl
		Organoarsenicals	DSMA, MSMA
		Others	Bromobutideflurenoldymron

4) Crop rotation:

Crop rotation also facilitates herbicide rotation. Many serious weeds are always associated with specific crops. Due to change of planting time in each crop, use of different weed control measures, the substituted crop can give the effective control of weeds.

Example:

Problem of *Phalaris minor*in rice-wheat system, could be tackled by inclusion of maize in kharifor berseem/sunflower/ mustard/barley or oat in rabi.

5) Tillage practices:

Minimum and zero till systems, the weed seeds remain close to soil surface and susceptible individuals are killed by herbicides and also the change of dilution of resistance from buried seed is less.

Deep tillage: Deep tillage/inversion tillage reduced the requirement of herbicides and delay the build up of resistance due to reduction in selection pressure.

OTHER CULTURAL PRACTICES TO AVOID OVER RELIANCE ON HERBICIDES

1) Stale seed bed preparation
2) Selection of competitive crop
3) Modifying the time of planting
4) Optimum seed rate
5) Crop stimulation

INTEGRATED WEED MANAGEMENT PRACTICES (FAO, 1966)

Adoption of IWM involving physical, cultural, chemical and biological methods in an integrated fashion without excessive reliance on any single method, can help in successfully managing herbicide resistance.

Example:

Summer deep ploughing + stale seed bed + use of weed free seed for sowing + post-emergence herbicides - can dilute the resistance against *Phalaris minor* in wheat.

References (if any)
1. **2.**

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