Chapter - 7
Perspective of Herbicide Resistance in Weeds and Their Management

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Abstract

Weeds have been a problem creator in crop production practices since time immemorial. The weeds cause around 37-45% crop yield reduction. Out of all the methods of control of weeds, the most prominently used one is the chemical control of weeds with the help of herbicides. The chemical control with herbicides being an effective and quick option towards weed control has made farmers more stooped towards them. The unavailability of labours and their high wages also aid in the increased usage of herbicides. This increased use of herbicides has led to the development and evolution of herbicide-resistant weeds, shift in weed flora and increased herbicide residues in the food chain and thus created an imbalance in the ecosystem. Herbicide resistance results from the evolution of weeds, resulting from the selection pressure laid by using similar herbicides or different herbicides having similar modes of action. Therefore, their management in crop production is as important as crop production itself. This chapter acknowledges the concept of herbicide resistance and its development, mechanism and types. It brings to light the technological options for the management of herbicide-resistant weeds. The management of herbicide resistance weeds does not rely on a single measure to control weeds but includes an array of techniques and tools to prevent the evolution and spread of these weeds. Thus, an appropriate combination of chemical, non-chemical, biotechnological and genetic methods would be greatly helpful in the management of herbicide resistance in weeds.

Keywords: Chemical control, herbicide resistance, management, mechanism, weed flora

Introduction

Weeds are plants that interfere the human activity. They Grow along with the plants and compete for water, nutrients, light and space. These

unwanted plants reduce the quality of agricultural produce and reducing farmer's net returns. The production cost also increases due to the measures adopted to control them. Losses in food grain production worth Rupees 3 lakh crore occur yearly due to insect pests, diseases and weeds. Among all the pests, weeds cause maximum yield losses (34%) than any other pest (Fig. 1).

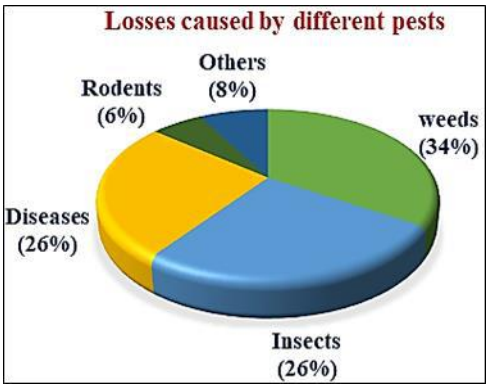

Fig 1: Yield losses caused by different pests

This necessitates the control of weeds through various methods, viz. preventive, cultural, mechanical, biological and chemical methods. Chemical control through herbicides has been the most prominent and irreplaceable method of weed control. The use of herbicides in optimum amounts has been reported to increase crop yields and reduce the cost of production, besides reducing the impact of heavy implements on the soil. Herbicides provide timely and effective weed control when combined with other methods. Many herbicides belonging to different chemical groups having different modes of action have been effectively used to control problematic weeds in different crops.

Along with the advantages of using herbicides, there are certain unignorable disadvantages, including a shift in weed flora, resistance in the weed species and ecosystem imbalance. With the beginning of chemical weed control through herbicides in the 1940s, herbicides have become a major driver of the green revolution. Since then, the use of herbicides has increased many folds. Currently, around 2 million tonnes of pesticides are used globally, with 47.5% being herbicides, 29.5% being insecticides, 17.5% being fungicides and 5.5% other pesticides. This overreliance on herbicides has led to the evolution of herbicide resistance in weeds. Herbicide resistance is a global problem, and the number of resistant weed biotypes is expanding at an alarming rate, which poses a serious concern to the farming community and weed scientists.

Herbicide resistance

Herbicide resistance is "the hereditary capacity of a weed to live and reproduce after exposure to a herbicide dose typically deadly to the wild type". It is the altered reaction of a weed species that were once vulnerable to the point where some members are no longer susceptible. Herbicide resistance is a site-specific, imparted by a single, major (i.e., big phenotypic effect) gene with high dominance. Herbicide resistance is commonly found at shallow levels in weed populations before the herbicide application. Variation exists throughout every group, with some able to withstand herbicide application. Herbicide resistance also defined as the “naturally occurring inheritable ability of some weed biotypes within a weed population to survive a herbicide treatment that should, under normal use conditions, effectively control that weed population”. A group of plants within a species known as a biotype share biological characteristics with one another that the rest of the population does not, such as herbicide resistance.

When a particular class or family of herbicides is repeatedly used to control a specific weed population, weed populations change in genetic composition such that the frequency of resistant individuals increases. Thus, the weed populations become adapted to the potent selection posed by the herbicides.

History of herbicide resistance

The herbicide resistance was initially reported in wild carrots (*Daucus carota*) in Hawaii against 2,4-D in 1957. However, Ryan was the first to confirm herbicide resistance in common groundsel (*Senecio vulgaris*) against triazine herbicide in the USA in 1970. The first survey of herbicide-resistant weeds was conducted by Homer Le Baron and reported in 1990 and 1991.

Since then, the number of herbicide-resistant weeds has increased many folds. The increase in herbicide-resistant weeds has witnessed exponential growth in the last three decades. There are currently 515 unique cases of herbicide-resistant weeds globally (Fig. 2A), with 267 species (154 dicots and 113 monocots). Weeds have evolved resistance to 21 out of the 31 known herbicide sites of action and to 165 different herbicides. Weeds resistant to herbicides have been found in 97 crops in 72 countries. The United States (128) has the most herbicide-resistant weeds, followed by Australia (89), Canada (54), China (40) and India (4). The world's worst herbicide-resistant weed is *Lolium rigidum* (12 nations, 14 sites of action, 9 cropping regimes, over 2 million hectares), followed by *Echinochloa crus-galli*, *Poa annua*. *Amaranthus palmeri, Avena fatua* (Fig. 2B).

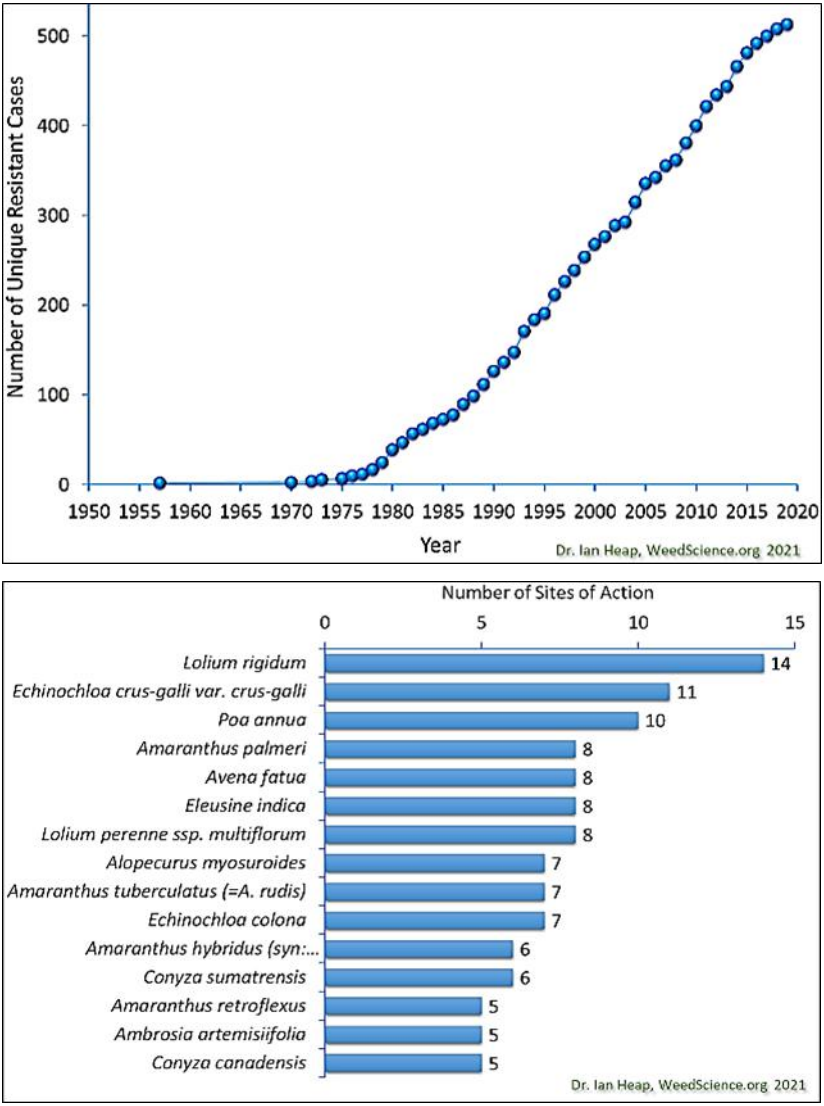

Fig 2A: The chronological increase in herbicide resistant weeds B. Graph showing most important herbicide resistant weed species

In India, the first case of herbicide resistance was observed in *Phalaris minor* in wheat against the herbicide isoproturon reported by Malik and Singh (1993). This was also the most severe case of herbicide resistance in the world.

Different herbicide sites of action have different propensities to develop resistance. The ALS inhibitors (HRAC 2) (162 resistant species) are most

prone to resistance, followed by PS-II inhibitors (HRAC 5) (103 species) and the ACCase inhibitors (HRAC 1) (48 species) (Fig. 3A). A maximum number of resistant weed species exists against atrazine (66), followed by glyphosate (51) (Fig. 3B).

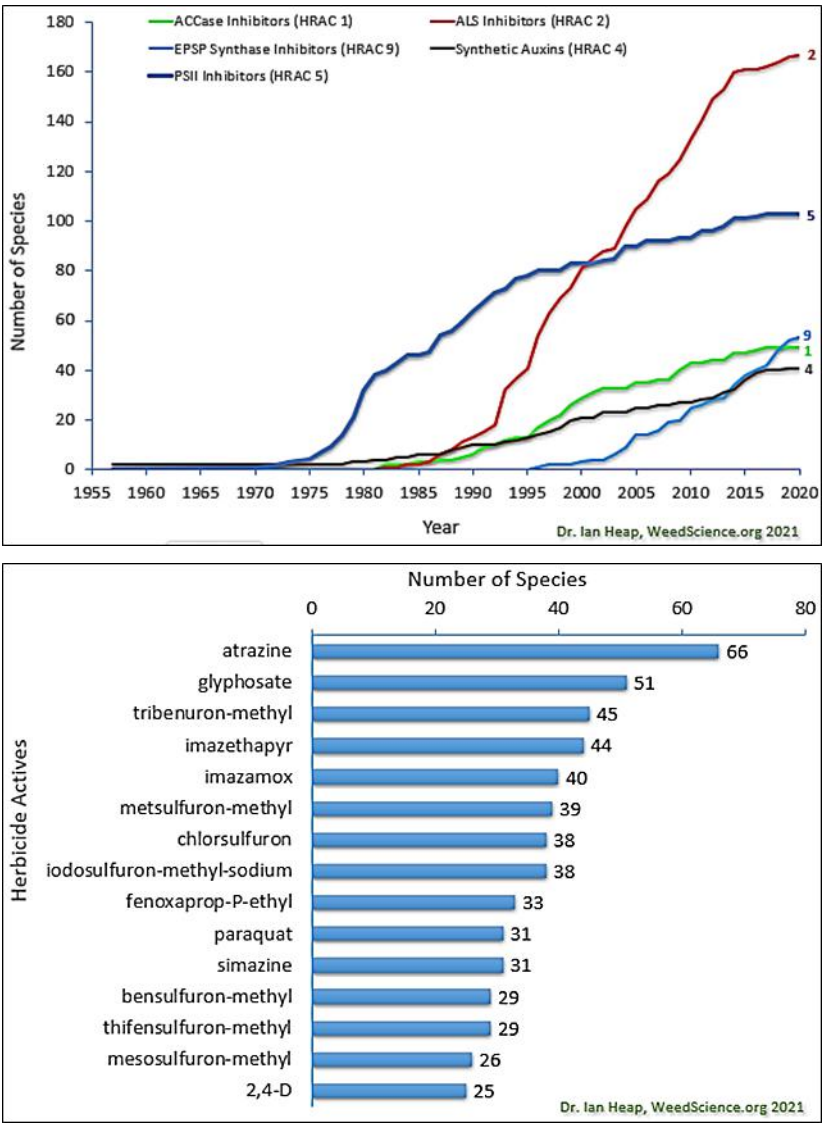

Fig 3A: Number of herbicide resistant weeds for several Herbicides Sites of Action (Herbicide Resistance Action Committee-HRAC Codes) B. Number of resistant species to individual active herbicides (Top 15)

Types of herbicide resistance

Herbicide resistance is a growing and unavoidable phenomenon. When herbicides with similar action modes are applied to control weed population, weeds develop one or the other kinds of resistance. Herbicide resistance can be classified depending on the mode of action, which is reflected by the biochemical process by which an active component affects the target plant.

Cross resistance

When resistance in a weed population occurs due to the continuous use of two or more herbicides having similar modes of action due to the presence of a single resistance mechanism (conferred by a single gene mutation) is called cross resistance. A weed biotype may evolve to become resistant to herbicide A after extensive use of herbicide A in a field. Still, it is also resistant to herbicide B, even though herbicide B was never used in that field owing to the similar mechanism of action of both herbicides. The sulfonylurea-resistant *Lolium rigidum* with a resistant ALS enzyme exhibit cross-resistance to imidazolinone and triazolopyrimidine herbicides (Saari *et al*., 1994) due to a similar mode of action (Acetolactate synthase inhibition). Common cocklebur resistant to one herbicide (chlorimuron) may also be resistant to other herbicides (imazaquin) due to ALS inhibition. One case of cross-resistance was reported from Australia, where a biotype of wild oats *Avena fatua* became resistant to fenoxaprop (an ACCase inhibitor, i.e., Acetyl Co enzyme-A Carboxylase), as well as several other ACCase inhibiting herbicides.

There are two broad categories of cross-resistance

a) Target site cross-resistance.

b) Non-target site/metabolic cross-resistance.

Target site cross-resistance: A target site is a region in a plant, generally an enzyme, where an herbicide's active ingredient interacts and interferes with physiological functions. Target site-based cross-resistance occurs when an alteration at the target site of one herbicide also confers resistance to herbicides from a different chemical class that inhibit the same target site in the plant. The sulfonylurea-resistant biotypes of *Lolium rigidum* with a resistant ALS (acetolactate synthase) enzyme exhibit varying levels of target site cross-resistance to the chemically distinct but ALS-inhibiting, imidazolinone and triazolopyrimidine herbicides change in the target site enzyme ALS which plays a vital role in the biosynthesis of branched-chain amino acids. Resistance of the wild oat species *Avena fatua* and *Avena*

sterilis to aryloxyphenoxy propionic acid and cyclohexanedione herbicides is endowed by the alteration in ACCase (acetyl-CoA carboxylase) enzyme preventing the lipid biosynthesis.

Non-target site/metabolic cross-resistance: Cross-resistance to different herbicide classes conferred by a similar process and similar rate of degradation of the herbicides is known as metabolic cross-resistance. A plant with non-target site resistance may metabolically detoxify an active herbicidal ingredient, reduce herbicide absorption or translocation, or sequester the herbicide in a cellular site that is not sensitive to the active ingredient. *Phalaris minor* showing resistance to isoproturon and diclofop-methyl. A study revealed that *Lolium rigidum* population resistant to diclofop-methyl also showed resistance to ALS inhibiting herbicide chlorsulfuron, which may be due to the enhanced metabolism of the herbicide.

Negative cross-resistance/collateral resistance

It refers to the phenomenon in which a weed biotype resistant to a chemical class of herbicide exhibits higher or increased susceptibility to other herbicides than its natural wild-type susceptible weed population. This generally happens due to changes in the target enzyme. For example, the triazine-resistant biotype of *Echinochloa crus-galli* was 53 times more resistant than the susceptible one, 33 and 2 times more sensitive to fluazifop-butyl and sethoxydim, respectively. The resistant weed biotypes of *Phalaris minor* can be more effectively controlled by fenoxaprop-p-ethyl than the wild susceptible type. Triazine-resistant weed biotypes of *Brassica napus* and *Amaranthus retroflexus* were more sensitive to bentazon.

Multiple resistance

The phenomenon in which a weed population develops resistance to two or more herbicides having different mechanisms of action is called multiple resistance. An individual plant (or a population) possesses two or more different resistance mechanisms that resist a single herbicide or different classes of herbicides. Multiple resistances can occur if a weed population resistant to an herbicide is treated with another herbicide frequently (without effective resistance management). The same weed population develops resistance to the second herbicide also, and so on. Multiple resistances can also emerge from pollen transfer (cross-pollination) between sexually compatible individuals harbouring various resistant genes. The amount of multiple resistances that can emerge is demonstrated by studies on one multiple-resistant population of *Lolium rigidum* VLR69. This population

was resistant to ACCase inhibitor herbicides (aryloxyphenoxy propionates) and ALS inhibitors (sulfonylurea and imidazolinone) herbicides. It had improved herbicide detoxification catalyzed by at least four separate Cytochrome P450s. It is now recognized that at least five genes are responsible for this population's high level of multiple resistance. Multiple resistance of common water hemp (*Amaranthus rudis* L.) to glyphosate and ALS-and PPO-inhibiting herbicides was confirmed in Missouri.

Reverse resistance

The phenomenon in which the weed biotypes resistant to a herbicide become sensitive to the same herbicide when, after not being used for 7-10 years, some alternate herbicides are employed to eliminate the resistant weed population.

Co-resistance/Compound resistance

A resistance phenomenon in which a weed develops resistance to both mixing partner herbicides of a mixer applied concurrently. *Lolium rigidum* developed resistance to amitrole and atrazine applied concurrently.

Causes for the development of herbicide resistance

Herbicide-resistant plants are initially in a weed population in minimal numbers (about 1 in 100,000 to more than 1 in 1,000,000). The repeated use of one herbicide, or herbicides that kill the plants the same way (same site of action or same herbicide group), allows these few plants to survive and reproduce. The population of resistant plants grows until the herbicide no longer effectively controls it.

Several variables may contribute to herbicide resistance development.

1) **Over-reliance on herbicides:** The over-dependency on herbicides for weed control due to labor shortages and increased wages pushed in the development of herbicide resistance.
2) **Continuous use of herbicides:** The continuous use of a class of herbicides with a similar mode of action is a significant factor in developing herbicide resistance in weeds.
3) **Seed bank in soil:** The development of herbicide resistance is more rapid whenever weed seed turnover in the soil is rapid. This turnover in the soil is influenced by cultural practices (ploughing).
4) **Monoculture:** Cultivation of a single crop every season favours the development of single or very few dominant weed species, which are generally controlled by the farmers using the same herbicides

every year, which not only confers herbicide resistance but is also responsible for weed flora shift.

5) **Tillage practices:** Zero/Minimum tillage favours the evolution of herbicide resistance as the susceptible weed species are killed, and the number of resistant weed seeds in the surface layer will increase following the exposure to similar herbicides. As seen in conventional tillage, there is no renewal of susceptible seeds from deeper soil layers or no burial of resistant seeds in a deeper layer.

Factors regulating the development of herbicide resistance

Factors typically responsible for developing herbicide resistance are vulnerable to selection pressure, which imposes resistance in plants. This selection pressure can be induced by the repeated application of the same herbicide, using a long residual soil-applied pre-emergence herbicide, or by the repeated application of the same post-emergence herbicide. Several aspects contribute to the development of herbicide resistance; however, the critical contributors are weed traits, herbicide chemical properties, and cultural practices, which are briefly reviewed here.

Weed characteristics

The most common weed characteristics that favour in development of herbicide resistance in weeds can be-

- **Initial frequency for resistant development:** Herbicide resistance appears from the selection of natural mutations that exist naturally as a small fraction of the population of resistant plants. This fraction usually occurs once in a million and is called initial frequency. In a natural weed population, if the initial frequency of the resistant individual is high, resistance will emerge more quickly than in a population with low frequency. Over time and seasons, such a small number of resistant plants continue to thrive and multiply each generation. These herbicide-resistant biotypes make a dominant weed population by killing the susceptible ones with similar herbicides. The initial frequencies for triazines, ALS inhibitors, and ACCase inhibitors are 1×10^{-7}, 1×10^{-3} and 1×10^{-3}, respectively.
- **Selection pressure:** When herbicides are applied to a weed population, susceptible individuals are killed, increasing the pressure for resistant ones to grow. Thus, selection pressure is defined as the relative proportion of live resistant and susceptible individuals that survive an herbicide treatment (Fig. 4). The

frequent application of similar herbicides having the same site and modes of action over several growing seasons without any rotation imposes high selection pressure.

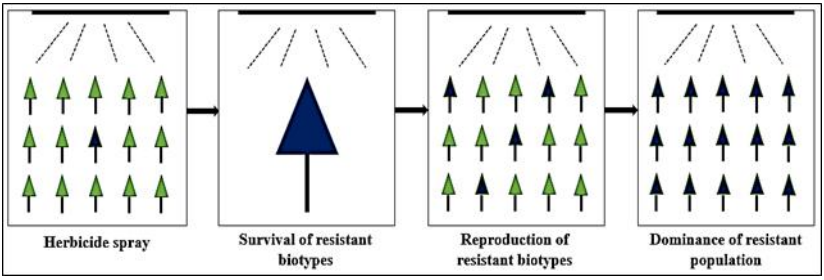

Fig 4: Selection pressure in weed population

- **Biological fitness:** It measures the potential evolutionary success of a genotype. If the biological fitness of a resistant weed biotype is more, it will quickly increase the rate of resistance development. If the biological fitness of a susceptible biotype is more, it will take more time for resistance development.
- **Seed bank in soil:** The reserve of all viable weed seeds and vegetative propagules present in the soil is known as seed bank. The weed species whose seeds remain in soil seed bank delays the herbicide resistance due to continuous germination of susceptible seeds from seed bank. The soil seed bank can have a significant buffering effect in delaying the emergence of resistance.

Herbicide characteristics

The continuous use of the same kind of herbicides as a primary method of weed control is a major factor in developing herbicide resistance in weeds. The herbicide characteristics also affect the rate of herbicide resistance in species.

- **Herbicides having very specific mode of action:** The application of herbicides having one target site imparts more selection pressure. Change at only one target site offers resistance in the biotype.
- **Type of herbicide and its time and dose of application:** The pre-emergence herbicides exert higher selection pressure as they are applied before the germination of weeds and are more efficient in killing weeds than post-emergence herbicides.
- **Prolonged residual activity:** Long residual activity in soil will eliminate susceptible biotypes or repressed for a more extended

period, allowing the resistant ones to flourish and reproduce in a competition-free environment.

- **Herbicide metabolism:** The herbicides which are metabolized quicker have less chance to develop resistance due to changes in herbicide at the site of action.

Mechanism of herbicide resistance

There can be two mechanisms by which resistance in weeds occurs-

Exclusionary resistance

The resistance mechanism excludes the herbicide molecules away from the site of action in the plant. As a result, the herbicide cannot concentrate at the site of action in sufficiently lethal quantity. The exclusion of herbicide molecules can take place in many ways. Plants develop resistance due to the inaccessibility of herbicide molecules at the site of toxic action. Exclusion of herbicide from the site of action can be due to several reasons:

a) **Differential nutrient uptake:** Herbicides are less readily absorbed in resistant biotypes due to morphological differences such as excessive wax production, decreased leaf area, and so on. Differential herbicide absorption can occur due to morphological barriers on leaves, such as a highly enhanced waxy layer on the cuticle, hairy epidermis, low leaf quantity and size.

b) **Differential translocation:** The less uptake of herbicide by the resistant biotypes generally results in less translocation of herbicides. In resistant biotypes, the apoplastic (cell wall, xylem) and symplastic (plasma lemma, phloem) translocation of herbicide is reduced. In *Lolium rigidum*, the resistant biotypes show less translocation of chlorsulfuron than susceptible ones.

c) **Sequestration and compartmentation:** It refers to the storing, accumulation and sequestration of herbicide in vacuoles, cells, or tissues away from the actual site of action of that herbicide and thus prevents the reaching of herbicide at the site of action. This means that the herbicide is removed from sensitive parts of the plant cell to a tolerant site, such as a vacuole, which is effectively harmless to plant growth. One of the key mechanisms of paraquat resistance is compartmentation in *Poa annua*. Sequestration is also observed in some resistant biotypes of *Lolium rigidum* in Australia.

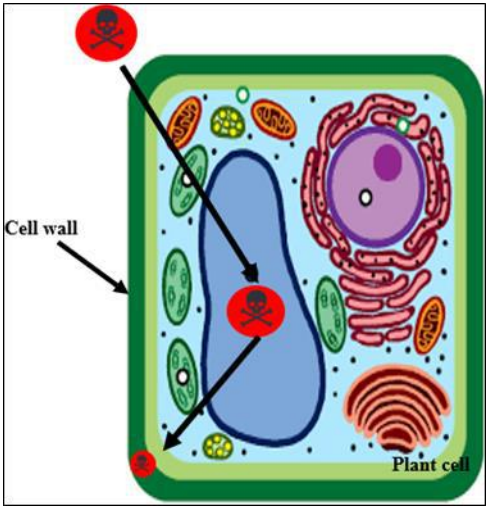

Fig 5: Herbicide compartmentation and sequestration

d) Enhanced metabolism/degradation: Quickly degrades herbicide molecules into non-toxic metabolites in the plant before reaching the site of action. Resistance to atrazine in some *Abutilon theophrasti* populations is due to the increased activity of glutathione-s-transferase, which detoxifies atrazine. Propanil resistance in *Echinochloa colona* is caused by increased activity of the enzyme aryl-acyl amidase, which detoxifies propanil. Resistance to ACCase, ALS and PSII inhibitors is caused by increased herbicide metabolism caused by cytochrome P450 monooxygenase in various grass weed species (*Phalaris minor*). Rapid herbicide breakdown and conjugation into non-toxic or less-toxic forms are fundamental resistance mechanisms in some weed species.

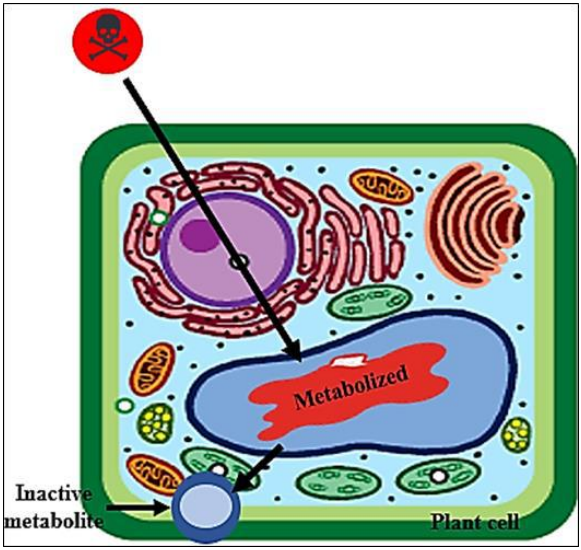

Fig 6: Enhanced metabolism of herbicide in resistant biotypes

The target site of action of resistance

a) **Altered site of action:** It is the alteration in the binding site of an herbicide due to some genetic changes in biotypes showing resistance, and thus herbicide molecule does not bind to site of action. This type of resistance is frequently accompanied with resistance via altered herbicide binding to its target protein (Fig. 7A). This is due to a single nucleotide alteration (mutation) in the gene that codes for the protein that the herbicide ordinarily binds to. This alters the amino acid sequence of the protein, reducing or eliminating the herbicide's capacity to interact with it. This hereditary change to herbicide sites of action is involved in the processes responsible for the majority of triazines, ALS inhibitors, and dinitroaniline herbicide resistance. Resistance of *Phalaris minor* to clodinafop occurs due to change in target site.

b) **Site of overproduction:** In this, the site of action (target protein) is enlarged or overproduced; as a result dilution effect of herbicide occurs. The site of action in an excess amount and so normal rate of herbicide application cannot deactivate the total enzyme produced (Fig. 7B). As a result, the additional enzyme generated by the plant biotype can allow it to go on with its usual metabolic functions, overcoming the herbicide's lethal effect.

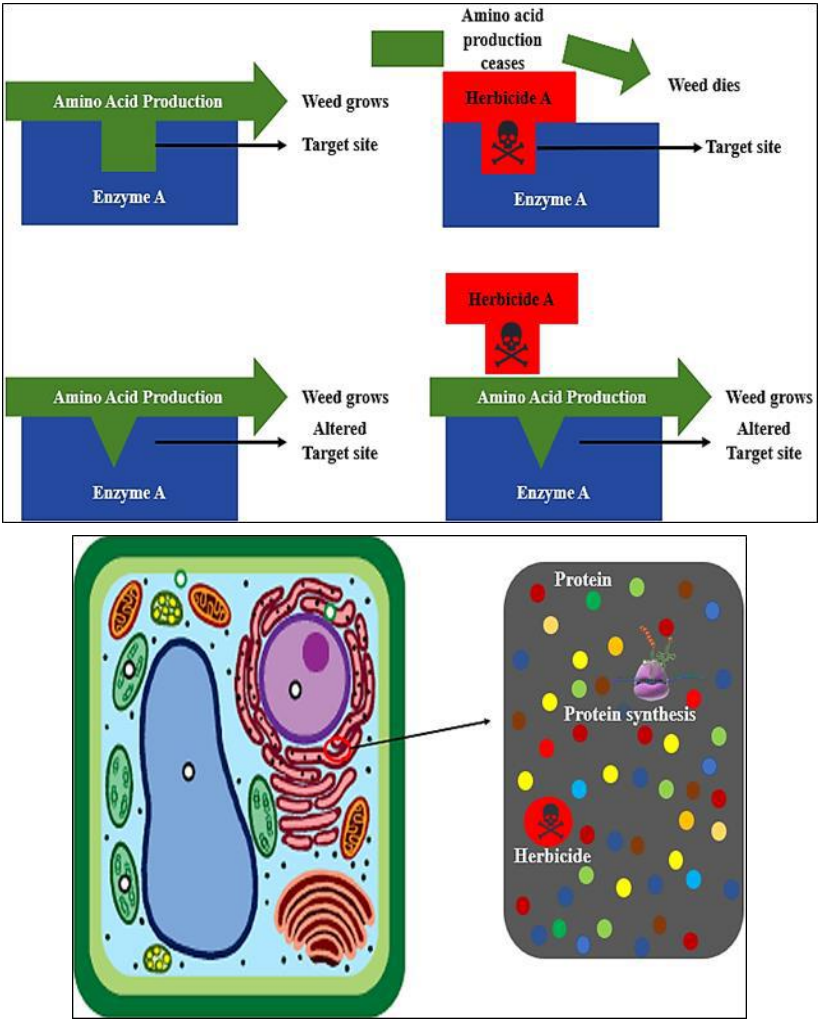

Fig 7: A. Altered site of action in weeds B. Overproduction of target proteins

Management of herbicide resistant weeds

It is nearly hard to restore susceptibility to its pre-resistance level in that group because the resistance is essentially irreversible. Therefore, it is critical to start controlling herbicide-resistant weeds as soon as they are detected; otherwise, it may be difficult to control weeds safely and efficiently. The main principle behind the management of herbicide resistance in weeds is the reduction of selection pressure which leads to the evolution of resistance. As a result, the primary focus should be on addressing those factors and practices contributing to the rapid emergence of resistance and managing them.

The various strategies for the management of herbicide-resistant weeds are:

Preventive methods

Any weed control method that reduces selection pressure for resistance will prevent resistance from emerging. Preventive methods aid in stopping the spread of weed seeds from one field to another.

Weed preventive approaches include:

- **Farmer's participatory approach:** Farmer's Awareness of the emerging resistance and participation in any weed management programme is essential. This strategy aids in limiting the physical spread of resistance to unaffected regions and developing suitable management procedures for resistance.
- **Prevention of seed rain:** Prevention of production and addition of new weed seeds in the soil and depletion of existing weed seeds in the soil seed bank reduces the selection pressure for herbicide resistance.
- Use of weed-free crop seeds.
- **Crop rotation:** Some weeds remain associated with specific crops. Crop rotation as a resistance management method is based on avoiding successive crops in the same field that require herbicides with the same site of action to control the same weed species. Crop rotation allows for adjusting planting time, weed infestation spectrum, production practices, herbicide mode of action, stage and application method. Using a combination of weed management tactics improves the chances of eradicating resistant biotypes and reducing the probability of their establishment. *Phalaris minor* can be effectively controlled in rice-wheat cropping systems by cultivating maize in *Kharif* or berseem in the rabi season for 3-4 years.
- Use clean farm implements in the fields since these are significant agents for dispersing weeds from one region to another.

Cultural methods

Any weed management strategy which aims to manipulate the field environment in favour of crops rather than weeds is known as a cultural method.

- **Selection of weed-competitive crop cultivars:** Cultivars which are naturally competitive to the weeds should be cultivated, which

smothers the weeds growing along with them and thus does not require any chemical control.

- **Stale seedbed technique:** One or two weed flushes are destroyed before crop planting wither through the application of non-selective herbicides or ploughing of the field 4-5 times before sowing crops.
- **Crop simulation:** The technique of giving selective favour to the crop in the form of fertilizers and irrigation is known as crop simulation. In this technique, irrigation provides to the crops in a row zone only.
- Closer row spacing.
- Timely sowing and rate of seeding.
- Good crop husbandry.

Mechanical methods

- **Tillage:** In minimum and zero till systems, the weed seeds remain close to soil surface and susceptible individuals are killed by herbicides and the change of dilution of resistance from buried seed is less. Zero/minimum tillage practices which depend on the use of herbicides to kill the weeds may aggravate the problem of herbicide development. However, Deep tillage through the inversion of soil brings the deep buried weed seeds on the surface continuously and thus, reduce the requirements of herbicides and delay the build of resistance due to reduction in selection pressure.

Herbicide management

- The use of herbicides only, when necessary, proves to be a sound method to delay herbicide resistance in weeds.
- Abandonment of pesticides to which weed has developed resistance.
- **Use of alternative herbicides:** Immediately after the development of resistance, the use of that herbicide should be stopped. Using alternative herbicides with different chemistry and mechanisms of action can be advised as a short-term remedy in HR weed management if cost-effective alternative herbicides are available. For example, Isoproturon-resistant *Phalaris minor* is more effectively controlled by pendimethalin, clodinafop, fenoxaprop and sulfosulfuron.

- **Herbicides selection:** Herbicides having a low risk of developing resistance should be applied instead of herbicides having a high risk. Despite widespread usage of these herbicides, few weeds have developed resistance to chloroacetamides, diphenyl ether and glyphosate. As a result, they are regarded as posing a minimal danger to the selection of herbicide-resistant weeds. On the other hand, Weeds quickly developed resistance to ALS inhibitors, triazines, bipyridylium, phenyl ureas and ACCase inhibitors.
- **Herbicides mixtures and rotation:** Using two or more herbicides having a different mode of action simultaneously in a field is called herbicide mixtures. Using different herbicides with different mechanisms of action in the subsequent seasons is called herbicide rotation. When two or more herbicides with distinct modes of action are used in a mixture or rotation, the selection pressure for resistance biotype is reduced, and the rate of evolution is slowed. Thus, the herbicides should be rotated once every three years. These herbicide mixtures are also effective against diverse weed flora. Different in the mechanism of action, they also reduce the chance of weed flora shift and the evolution of herbicide resistance in weeds. *Xanthium strumarium* can be controlled by imazaquin in one year and atrazine in the other.
- **Site-specific herbicides application:** Site-specific management using a global positioning system can be beneficial in monitoring and managing HR weed patches in a field at an early stage of growth throughout time. Site-specific herbicide treatment, which would use weed abundance to delineate application regions in an area, might allow for some reduction in overall selection pressure, thereby reducing herbicide resistance development.

Integrated weed management

The holistic approach of weed management involving all methods of weed control, viz. preventive, cultural, mechanical, biological and chemical methods in an integrated manner to keep the weed population below the threshold density, can be successfully used in the management of herbicide-resistant weeds. Summer deep ploughing + stale seedbed technique + use of weed-free seed during sowing + post-emergence herbicides can dilute the resistance in *Phalaris minor* in wheat. IWM practices are the most critical and exclusive method for successfully controlling multiple resistance in weeds.

Newer approaches

Herbicide-resistant crops: Adoption of genetically modified herbicide-resistant crops is generally motivated by simpler and improved weed management or greater net returns. The judicious use of HR crops can inhibit the selection of HR weeds by enhancing herbicide rotation alternatives, such as substituting high-risk herbicides with lower-risk ones. However, the repeated use of HR crops in cropping systems, which results in the reapplication of herbicides with the same mode of action, may select for newer HR weed biotypes.

RNAi technology: The use of RNA interference (RNAi) technology is another emerging approach that has the potential to battle HR weeds. It is a unique technique of targeting certain genes, turning them off, and destroying specific RNA sequences, which prevent the production of particular proteins inducing herbicide resistance. Weeds resistant to ALS-, HPPD-, and PPO-inhibiting herbicides have also been used to illustrate the technology. However, this technology needs commercialization in emerging times.

Conclusion

Herbicide Resistance is an irrevocable phenomenon. Farmer's on-going and hesitant use of herbicides as a technique of chemical weed control has resulted in the development of herbicide resistance in weeds. This resistance develops due to the selection pressure on the few surviving resistant biotypes of weeds. Multiple resistances in weeds and a decline in the discovery of novel herbicide modes of action will pose the greatest threat to long-term weed control in agronomic crops in the coming years. The discovery of new herbicide sites of action and herbicide-resistant crop traits will play a significant role in weed control in the future. Still, growers must transition to integrated weed management, which employs all economically available weed control techniques. Then and only then can the ever-increasing problem of herbicide resistance in weeds be prevented.

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