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Herbicide resistance and its management

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Introduction

Weeds infest crops and reduce the harvestable yield. Almost all the crop production systems suffer from infestation of weeds. The weed flora of crops and cropping systems include grasses, broad leaved weeds and sedges. These could be annual, biennial and perennial. Growers adopt different management techniques to manage weeds. Those include manual or mechanical weeding, cultural operations and chemical methods. Use of herbicides has been the most effective method to manage weeds. A number of herbicides belonging to different chemical groups with different mechanisms of actions have been effectively used to control most of the problematic weeds of different crops. However, prolonged use of herbicides has resulted in the development of herbicide resistance which is a worldwide problem and a challenge for both the farming community as well as weed researchers.

Herbicide resistance is defined as the inherited ability of a weed or crop biotype to survive and reproduce following treatment with a dose of herbicide to which the original population was susceptible.[1] The earliest observations of resistance was in wild carrot to the herbicide 2, 4-D. The first confirmed report of herbicide resistance was in common groundsel against triazine herbicides. Over the years, the magnitude of resistance has gone up worldwide. These have been reported from 66 countries across the globe. A total of 250 species which involves 145 dicots and 105 monocots, have developed resistance to 160 different herbicides with different sites of action.[2] Repeated and intensive use of herbicides with similar mechanisms of action in crops/cropping systems over a period of time leads to development of resistant biotypes within the community. Gradually the resistant biotypes develop multiple resistance posing a greater threat to the production systems. Among the weed flora, the most important herbicide resistant species reported across the globe include *Lolium rigidium, Avena fatua, Amaranthus retroflexus, Chenopodium album, Elusine indica, Echinocloa cruss-galli*, and *Phalaris minor*.

Which factors influence resistance development?

The development of resistance is a result of a combination of factors that include biology of weed species, herbicides in use and operational methods. Overreliance on the use of herbicides including use of herbicides with a single site of action as well as multiple applications during the growing season, use of herbicides for consecutive growing seasons and herbicides that have long soil residual activity is the major cause of resistance development. Depending on the selection intensity, the resistant weed population will continue to grow and expand. Higher seed production capacity and efficient seed dispersal mechanism are some of the weed traits that helps spread resistance. The resistant biotypes may develop cross resistance[3] (resistant to two or more herbicides having the same mode of action) or multiple resistance (resistant to two or more herbicides having different modes of action) over a period of time. At the biochemical level, differential uptake, translocation and metabolism of the herbicide decides the fate of resistance development. Faulty management methods that rely upon single method of weed control, faulty quarantine systems, crop seed lots contaminated with seeds of resistant weed also contribute to the problem.

Mechanisms of resistance

Broadly, two categories of mechanisms operate. The first one involves target site resistance that includes alterations in the herbicide binding site and over-production of the target site. The second category includes non-target site resistance where reduced uptake and enhanced metabolism of the herbicide as well as its sequestration leads to development of resistance. Target site resistances include amino acid substitutions in or around the binding site as in case of AC Case and ALS inhibitors. Most widespread cases of resistances include resistance to inhibitors of photo systems I and II, acetolactate synthase (ALS), acetyl-CoA carboxylase (AC Case), protoporphyrinogen oxidase (PPO), carotenoid synthesis, EPSP synthase and mitosis inhibitors. Resistance may also occur due to limitations in translocation of the herbicide molecules to the target site due to their degradation by enzymes. One such group of enzymes include the cytochrome P450 monooxygenases. Resistance could also be due to more than one mechanism operating simultaneously.

Resistance to Photo system I and II inhibitors are most widespread. Triazines, Triazinones, Uracils, Nitriles, Phenylureas, Pyridazinones and Benzothiadiazole groups include the PS II inhibitors. About 67 weed species have been reported to be resistant to triazines. A mutation of the D1 protein resulting in a Serine 264 to Glycine change has been established as the mechanism of resistance. Non-target site based mechanisms such as enhanced metabolism either by glutathione-s-transferase or cytochrome P450 has also been proposed. Photo system I inhibitors such asparaquat and diquat were being used for non-selective weed control around the world. Tolerance to paraquat was first reported in *Lolium perenne* followed by *Amaranthus lividus, Bidens pilosa, Eleusine indica* and *Solanum nigrum*. The mechanism for paraquat resistance has been ascribed to decreased herbicide translocation. Resistance to the Aryloxyphenoxy propionates (AOPP) and Cyclohexanediones (CHD) has become widespread particularly in *Lolium rigidium* in Australia and in *L multiflorum, A fatua* and *Setaria viridis* in N America. Other weed species resistant to this group include *Digitaria sangunalis*, *Echinochloa crusgali* and *E. Colona.* An aspartate to glycine change in the carboxyl transferase domain of AC Case and non-target site mechanisms have been proposed for resistance in *Lolium multiflorum*. Recent reports of

P minor resistance to this group have been ascribed to target site modifications with mutations in one or more gene encoding the AC Case enzyme Gherekhloo et al.[4] Sulfonylureas, Imidazolinones, Triazolopyrimidines, and pyrimidinyl (thio) benzoate primarily inhibit the enzyme acetolactate synthase. Resistance to this group was first noticed in *Lactuca serriola*. Other resistant species include *Amaranthus sp.*, *Avena fatua*, *Eleusine indica* and *Lolium sp.* Mostly target site-mediated resistance mechanism have been proposed which includes nucleotide substitution of a G with a T.

Glyphosate has been a potent herbicide which is applied post-emergence. It kills plants by inhibiting the EPSP synthase, a key enzyme in the shikimate pathway for the biosynthesis of aromatic amino acids. Resistance to this herbicide started appearing in 1994. By now, glyphosate resistance has been reported in 16 weed species in 14 different countries and is becoming a significant problem worldwide. Both target site EPSSPS gene mutation/amplification ad non-target site resistance mechanisms have been proposed. Italian ryegrass (*L multiflorum*), Goose grass (*Eleusine indica* L), horseweed (*Conyza canadensis L*) are some of the species reported to be resistant to this herbicide. The physiological mechanism of resistance to glyphosate has been proposed as dissimilar mobility of the herbicide in the whole plant in resistant and susceptible biotypes with less downward translocation in the R biotype in horseweed. The tolerance mechanism in velvetleaf has been reported to be associated with the differential disruption of cellular processes in source leaves and sink tissue. Resistance to tubulin assembly-inhibiting herbicides such as dinitroanilines, benzoic acids, pyridines and carbamates that target germinating seedlings have been reported in only 10 weed species. Both target site and non-target site mechanisms exist. Resistance to carotenoid biosynthesis inhibitors have been reported in *Hydrilla verticillata* in USA and *Raphanus raphanistrum L.* in Australia. However, low incidence of resistance observed in this group makes them attractive herbicide tools for the future. Herbicides that inhibit protoporphyrinogen oxidase (protox or PPO) are used widely to control weeds in a variety of crops. They pose a low risk for selection of HR weed biotypes.

Phalaris minor resistance to isoproturon is one of the most serious cases of herbicide resistance in the world. Resistance in this weed was reported from wheat fields where isoproturon was used for over 10years.[5] Over the years, this species has developed multiple resistances to herbicides with different mechanisms of action such as ALS inhibitors, AC Case inhibitors, and premix of herbicides mesosulfuron and idosulfuron.[6] Both target site and enhanced metabolism are reported from Haryana while resistance due to enhanced metabolism has also been reported from Uttarakhand, India.[7] This poses a major threat to wheat growers in India and a major challenge to the researchers.

Management of herbicide resistance

Resistance to herbicides is a serious problem worldwide. The cases of resistance are increasing at an alarming rate. Cross and multiple resistances have further complicated the situation. Though arrival of new molecules with different mechanisms of action can help overcome present cases of resistance, an overreliance on chemical weed control will continue to give rise to cases of resistance. Therefore, alternate management strategies must be developed to combat resistance. Resistance management requires both preventive as well as reactive approaches. Prevention will include suitable combinations of weed management methods such as cultivation practices, crop rotations, field scouting and herbicide rotations. Seed production by suspected resistant biotype should be checked to prevent spread of resistance to other areas.[8]

Crop production programmes worldwide have been focussing on techniques and issues such as conservation tillage, sustainability of resources, resource use efficiency and most important of all, the changes in global climate. Therefore, integrated approaches involving judicious combinations of cultural, mechanical, biological and crop and herbicide rotations must be adopted to reduce the dependence on herbicides. Tillage method, planting time, method of herbicide application, optimum dose, stale seed bed and zero tillage are some of the short duration resistance management strategies. Allelopathic cultivar development could be another strategy to mitigate the herbicide load. Rapid resistant screening techniques should be developed which requires simple methods yielding quick results. Physiological and biochemical studies will be helpful to develop such screening techniques.

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Conflict of interest

Author declares that there is no conflict of interest.

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