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New techniques for herbicide resistance management in weeds: A review

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Abstract
Herbicides are most effective and economic tool among the weed management practices. Use of herbicide is rapidly increasing in the world including India. Along with the advantages there are some disadvantages like herbicide resistance and environmental concern due to repeated use of herbicide. Development of resistance against the herbicides in targeted species is the most prominent among them. Herbicide resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. Herbicide resistance is a worldwide phenomenon and number of resistant biotypes of weeds is increasing at an alarming rate. There are currently 480 unique cases of herbicide resistance globally in 252 plant species. Herbicide resistance in weeds is a product of evolution in cultivated fields, responding to the selection pressure laid by the use of such herbicides. Continuous use of the same herbicide or herbicides having same mechanism of action in mono culture with minimum tillage has been the major causes of occurrence for herbicide resistance. Altered site of action, enhanced metabolism, over-expression of the target protein and sequestration are the main mechanisms of herbicide resistance in weeds. Herbicide resistance problem can be addressed with integrated weed management approaches, including crop and herbicide rotations, herbicide combinations along with cultural, mechanical and biological methods.

Keywords: Herbicide resistance, plant resistance to herbicides, resistance managements, weeds resistance to herbicides

Introduction

Environmental stress on plant occurs when the level of an environmental condition or the availability of environmental resources adversely affects plant growth. Biodiversity is a product of evolution and natural selection. Plants being directly exposed to external environment are vulnerable to variety of stresses, therefore many plants, particularly weeds; contain enormous genetic potential to survive under such variations. Most weed species contain adequate genetic variations that allow them to survive under variety of environmental stresses. The ability of living organism to compensate for or adapt to adverse or changing environmental conditions is remarkable. In recent years, there has been an increasing reliance on modern herbicides leading to a reduction in the need for 'traditional' techniques of weed control. Cropping patterns have adapted, driven by the possibility to further increase crop output, to rely more and more on these products. While economically this shift has been rewarding to farmers, some negative consequences have emerged which now need to be addressed in the interest of longer term sustainability. One result of modern agriculture and the reliance on herbicides is the emergence of populations of weeds which are resistant to products designed to control them. All natural weed populations regardless of the application of any weed killer probably contain individual plants (biotypes) which are resistant to herbicides. Repeated use of a herbicide will expose the weed population to a "selection pressure" which may lead to an increase in the number of surviving resistant individuals in the population. As a consequence, the resistant weed population may increase to the point that adequate weed control cannot be achieved by the application of that herbicide. Herbicide resistance is "the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type". In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering or selection of variants produced by tissue culture or mutagenesis (WSSA, 1998) [12].

Defined herbicide resistance as "naturally occurring inheritable ability of some weed biotypes within a given weed population to survive an herbicide treatment that should under normal use conditions, effectively control that weed population" (HRAC, 2015).

Types of herbicide resistance

1. Herbicides cross resistance

When weeds are resistance to two or more herbicides resulting from the presence of single resistance mechanism is termed as cross resistance. Even new herbicides may offer new solution there may be resistance to them from the first time they are used. The presence of such a mechanism can complicate the selection of alternate herbicides as tools to control a resistance situation. If evolution of resistance to one herbicide immediately endowed resistance to other herbicides, there is cross-resistance. It is metabolic cross resistance if the herbicides or their toxic products are degraded by the same mechanism.

2. Multiple resistances

Multiple resistances is the phenomenon of resistances to herbicides from more than one chemical classes to which a population has been exposed. It refers to a weed or crop biotype that has evolved mechanisms of resistance to more than one herbicide and the resistance was brought about by separate selection processes. Multiple resistances were first reported in Lolium rigidum in Australia and Alopecurus myosuroides in Europe. Both the weeds are resistance to a large number of herbicides available to the cultivators in those countries. Herbicide resistant weeds are a global and growing problem (e.g. number of cases, resistant species, *etc*.). Although herbicide resistance was reported as early as 1957 against 2, 4-D from Hawaii (Bhatti *et al*., 2013) [1].

The first report of herbicide resistance was confirmed in triazine herbicide resistant common groundsel. Since then, in the last four decades, there has been many reports confirming resistance to other herbicides. There are currently 480 unique cases (species × site of action) of herbicide resistance globally in 252 species (145 dicots and 105 monocots). Weeds have developed resistance to 23 of the 26 known herbicide sites of action and to 161 different herbicides (Heap, 2017) [4].

Most of the herbicide resistance cases had observed in developed countries like U.S.A, Canada, Mexico, France, Spean, U.K and Australia. In India little seed canaq grass (Phalaris minor) has evolved multiple herbicide resistance (MHR) across three modes of action: photosynthesis at photosystem II, acetyl-coA-carboxylase (ACCase) and ALS inhibition. The MHR population had a low level of sulfosulfuron resistance but high level of resistance to clodinafop and fenoxaprop. Some of the resistant populations have GR50 values for clodinafop 12 times higher than susceptible population. The multiple herbicide resistant populations (resistant to sulfosulfuron, clodinafop, pinoxaden and isoproturon) are susceptible to the triazine (metribuzin and terbutryn) and dinitroaniline (pendimethalin and trifluralin) herbicides. Triazine herbicides have selectivity problem in wheat and due to lack of knowledge and non-availability of effective herbicides many farmers are facing severe yield losses due to multiple herbicide resistance (Rajender, 2014) [10].

Mechanisms of herbicide resistance

Mechanisms of herbicide resistance can be broadly grouped into two categories:

1. Target site resistance
2. Non-target site resistance

Herbicide resistance generally includes diverse mechanisms that utilize changes in biochemical processes within weed plants, for example, changes to exterior structures, and changes to germination period. Target site mutation and enhanced metabolism in non-target site resistance are the most commonly encountered mechanisms (Kwon *et al*., 2015) [7].

Target-site resistance occurs when the target enzyme of an herbicide becomes less sensitive or insensitive to the herbicide. The loss of sensitivity is usually associated with a gene-coding mutation for a protein, which can lead to conformational changes in the structure of the protein. The physiological changes can impair the ability of herbicides to attach to the specific binding site of the enzyme, reducing or eliminating the herbicidal activity. In short, target site resistance refers to a structural change to the binding site of herbicide molecule to confer resistance or when the target site is over expressed through gene amplification (Delye *et al*., 2013) [3].

(A) Altered site of action

Site of action is altered in such a way that it is no longer susceptible to the herbicide e.g. In Lactuca sativa biotypes which are resistant to sulfonylurea herbicides, the ALS enzyme which is the site of action of herbicide is modified in such a way that herbicide can no longer bind with the enzyme and inactivate it.

(B) Overproduction of site of action

It also happens in some cases that the site of action is enlarged or overproduced as a result dilution effect of herbicide occurs. The applied normal rate of herbicide is unable to inactivate the entire amount of enzyme protein produced. Therefore, the extra amount of enzyme produced by the plant biotype can allow it carry on its normal metabolic activities surmounting the lethal effect of the herbicide. Non target site resistance is associated with physiological mechanisms aimed at reducing the amount of herbicide reaching the target site (Im *et al*., 2016) [6].

These mechanisms mainly consist of decreased rates of herbicide penetration and herbicide translocation, and also an increased rate of herbicide sequestration or metabolism. Enhanced metabolization of herbicidal compounds enables the majority of non-target site resistance cases (Powles and Yu, 2010) [9].

(A) Differential herbicide uptake

In resistant biotypes the herbicides are not taken up readily due to morphological uniqueness like over production of waxes, reduced leaf area *etc*. It can be differential herbicide uptake due to the morphological barrier on leaves such as extraordinarily increased waxy coating on the cuticle, hairy epidermis and low foliage number and size *etc*.

(B) Differential translocation

In resistant biotypes the apoplastic (cell wall, xylem) and symplastic (plasma lemma, phloem) transport of herbicide is reduced due to different modifications. It can also be due to differential translocation whereby apoplastic (xylem tubes) or symplastic path (phloem cells) restrict or delay movement of right concentration of herbicide at the site of action (Ozair *et al*. 1987) [8].

(C) Sequestration and compartmentation

Compartmentation may be either by storage of the herbicide or its metabolites in the cell vacuole or their sequestration in cells or tissue, far from the site of action. One of the major mechanisms of resistance to paraquat is compartmentation, though alternative explanations such as rapid enzyme detoxification have also been suggested. Similarly sequestration is also found in some resistant biotype of L. rigidium in Australia (Tharayil and Santhakumar, 2003) [11].

(D) Metabolic detoxification

Herbicide is detoxified before it reaches the site of action at a rate sufficiently rapid that the plant is not killed. The biochemical process that detoxifies herbicides can be grouped into four major categories: oxidation, reduction, hydrolysis, and conjugation. Three enzyme systems are known to be involved in resistance due to increased herbicide detoxification. Resistance to atrazine in some population of Abutilion theophrasti is due to increased activity of glutathione-s-transferase that detoxifies atrazine. Resistance to propanil in Echinochloa colona is due to the increased activity of enzyme aryl-acyl amidase that detoxifies propanil. Increased herbicide metabolism due to cytochrome P450 monoxygenase is responsible for resistance to inhibitors of ACCase, ALS and PS-II in a number of grass weed species. Rapid degradation and or conjugation of herbicides into non-toxic or less-toxic form are major mechanisms of resistance in several weed species. There are several factors to consider when evaluating herbicide resistance risk. Some of these relate to the biology of the weed species in question, others relate to particular farming practices. Some examples are given below:

(A) Biology and genetic makeup of the weed species in question

1. Number or density of weeds

As resistant plant are assumed to be present in all natural weed populations, the higher the density of weeds, the higher the chance that some resistant individuals will be present.

2. Natural frequency of resistant plants in the population

Some weed species have a higher propensity toward resistance development; this relates to genetic diversity within the species and, in practical terms, refers to the frequency of resistant individuals within the natural population.

3. Seed soil dormancy potential

Plant species with an longer soil dormancy will tend to exhibit a slower resistance development under a selection pressure as the germination of new, susceptible, plants will tend to dilute the resistant population.

(B) Crop management practices which may enhance resistance development

1. Frequent use of herbicides with a similar site of action

The combination of 'frequent use' and 'similar site of action' is the single most important factor in the development of herbicide resistance. Cropping rotations with reliance primarily on herbicides for weed control: The crop rotation is important in that it will determine the frequency and type of herbicide able to be applied. It is also the major factor in the selection of non-chemical weed control options. Additionally, the cropping period for the various crops will have a strong impact on the weed flora present. Lack of non-chemical weed control practices: Cultural, mechanical and biological or non-chemical weed control techniques, incorporated into an integrated approach is essential to the development of a sustainable crop management system. If the chance of resistance development is high, there must be some guideline to manage it.

2. The prevention and management of herbicide resistance

The prevention of resistance occurring is an easier and cheaper option than managing a confirmed resistance situation. Experience has shown that simply changing herbicides is not enough to overcome resistance in the mid to long term and that a sustainable, integrated system needs to be developed which is appropriate for the farm in question. Integrated Weed Management is defined as the use of a range of control techniques, embracing physical, chemical and biological methods in an integrated fashion without excessive reliance on any one method.

Rotation of crops

The principle of crop rotation as a resistance management tool is to avoid successive crops in the same field which require herbicides with the same site of action for control of the same weed species. Crop rotation allows the following options: 1) Different crops will allow rotation of herbicides having a different site of action 2) The growth season of the weed can be avoided or disrupted 3) Crops with differing sowing times and different seedbed preparation can lead to a variety of cultural techniques being employed to manage a particular weed problem and 4) Crops also differ in their inherent competitiveness against weeds. A strongly competitive crop will have a better chance to restrict weed seed production.

Cultural Techniques

Cultural (or non-chemical) weed control methods do not exert a chemical selection pressure and assist greatly in reducing the soil seed bank. Cultural techniques must be incorporated into the general agronomy of the crop and other weed control strategies. Not all of the examples given are adequate in all situations. Some of the cultural measures for weed control could include: a) Cultivation or ploughing prior to sowing to control emerged plants and to bury non germinated seed b) Delaying planting so that initial weed flushes can be controlled with a nonselective herbicide c) Using certified crop seed free of weed d) Post-harvest grazing, where practical e) Stubble burning, where allowed, can limit weed seed fertility & f) In extreme cases of confirmed resistance, fields can be cut for hay or silage to prevent weed seed set.

Herbicide rotation and herbicide mixtures

Herbicide rotation or mixtures refers to the rotation or mixtures of Herbicide Site of Action against any identified weed species. HRAC has recently prepared a classification of herbicides according to site of action. When planning a weed control program, products should be chosen from different site of action groups to control the same weed either in successive applications or in mixtures. A general guideline for the rotation of chemical groups should consider:

A. Avoid continued use of the same herbicide or herbicides having the same site of action in the same field, unless it is integrated with other weed control practices.
B. Limit the number of applications of a single herbicide or herbicides having the same site of action in a single growing season.

C. Use mixtures or sequential treatments of herbicides having a different site of action but which are active on the same target weeds.
D. Use non-selective herbicides to control early flushes of weeds (prior to crop emergence) and/or weed escapes.

The use of chemical mixtures to prevent resistance

Mixtures can be a useful tool in managing or preventing the establishment of resistant weeds. For chemical mixtures to be effective, they should: 1) Include active ingredients which both give high levels of control of the target weed and 2) Include active ingredients from different site of action groupings.

The use of bioherbicide to prevent or manage resistance

Boyette *et al*. (2014) [2] studied that interaction of the bioherbicide Myrothecium verrucaria and glyphosate for Kudzu control. Kudzu is an exotic invasive weed in the southeastern U.S. that is difficult to control with current commercial herbicides. Some success for its control has been achieved using a bioherbicidal agent, Myrothecium verrucaria (MV). Spore and mycelial formulations of MV were tested alone and in combination with glyphosate for control of kudzu (Pueraria lobata) under greenhouse and field conditions in naturally-infested areas.

Additional to the above guideline, the grower should

A. Know which weeds infest his field or non-crop area and where possible, tailor his weed control program according to weed densities and/or economic thresholds.
B. Follow label use instructions carefully; this especially includes recommended use rates and application timing for the weeds to be controlled.
C. Routinely monitor results of herbicide applications, being aware of any trends or changes in the weed populations present.
D. Maintain detailed field records so that cropping and herbicide history is known.
E. What to do in cases of confirmed herbicide resistance.

In cases where a control failure has been confirmed as resistant, immediate action is required to limit further seed production of the resistant plants. The degree of the action will depend on the stage of the crop in the field and the extent of the problem like a) Eradicate the remaining weed population in order to limit build-up and spread of seed in the soil b) Limit the field to field movement of resistant populations by cleaning planting, cultivation and harvesting equipment to avoid transfer of resistant weed seed c) Avoid using the herbicide to which resistance has been confirmed unless used in conjunction with herbicides having a different site of action, active on the resistant weed population d) If the resistant population is widespread, consider grazing the crop or cut for feed being careful not to transfer resistant seed via manure e) Select these fields for rotation or set aside for the following cropping season and f) Seek advice to assist in the long term planning of weed control in these fields.

Conclusion

Herbicide resistance is worldwide phenomenon and number of resistant biotypes of weeds is increasing at an alarming rate. As the use of very efficient and highly specific with single site of action herbicides is increasing worldwide there will be more complicated situation of herbicide resistance. Continuous use of the same herbicide or herbicides having same mechanism of action in mono culture with minimum tillage has been the major causes of occurrence of herbicide resistance. Herbicide per se does not cause any mutation resulting herbicide resistance. Weeds with an diverse genetic background might have resistant biotype within a large population. Repeated use of same herbicide over several seasons in a same area exerts selection pressure on resistant individual to evolve. Altered site of action, enhanced metabolism and sequestration or compartmentation are the main mechanisms of herbicide resistance in weeds. Over-reliance on herbicide should be minimized and herbicide should be used integrated with other practices. Herbicide should be used in sequence or in rotation or as mixture. We must keep available all other alternative tools we ever had, including the manual, cultural, biological and other practices which should be used in an integrated manner.

Table 1: Assessment of the risk of resistance development per target species

Management options	Risk of resistance		
	Low	Moderate	High
Herbicide mix or rotation	>2 modes of action	2 modes of action	1 modes of action
Weed control in cropping system	Cultural, mechanical and chemical	Cultural and chemical	Chemical only
Use of same mode of action herbicide	1 year	More than one	>3 year
Cropping system	Full rotation	Limited rotation	No rotation
Weed infestation	Low	Moderate	High
Herbicide residual period	Short	Intermediate	Long

Reference

1. Bhatti KH, Parveen T, Farooq A, Nawaz K, Hussain K, Siddiqui EH *et al*. A critical review on herbicide resistance in plants. World Applied Sciences Journal. 2013; 27:1027-1036.
2. Boyette CD, Hoagland RE. Bioherbicidal potential of Xanthomonas campestris for controlling Conyza canadensis. Biocontrol Science and Technology. 2015; 25(2):229-237.
3. Delye C, Jasieniuk M, Lecorre V. Deciphering the evolution of herbicide resistance in weeds. Trends Gene. 2013; 29:649-658.
4. Heap IM. International survey of herbicide resistant weeds. Accessed in http://www.weedscience.org on, 2016.
5. http://hracglobal.com/files/Management-of-Herbicide-Resistance.pdf
6. Im SB, Lee SH, Kim YY, Kim JS, Kim DS. Construction of a full-length cDNA library from Cardamine manshurica Nakai and characterization of EST dataset. Korean Journal of Agricultural Science. 2016; 43:33-39.
7. Kwon YS, Choi IH, Kim CW, Choi MS, Kwak JH. Yield change of seed bulb according to annual field culture after induced meristem culture in garlic (*Allium sativum* L.). Korean Journal of Agricultural Science. 2015; 42:299-304.
8. Ozair CA, Moshier LJ, Werner GM. Absorption, translocation, and metabolism of foliage-applied chloramben in velvetleaf (*Abutilion theophrasti*) and

soybean (*Glycine max*). Weed Science. 1987; 35:757-762.
9. Powles SB, Yu Q. Evolution in action: Plants resistant to herbicides. Annual Review of Plant Biology. 2010; 61:317-347.
10. Rajender S, Chhokar Hari Virender. Integrated weed management in wheat.
11. Tharayil, Santhakumar N. Mechanism of Herbicide Resistance in Weeds. On line book Mechanism of Herbicides Resistance in Weeds. Weed Science Society of America, 2003. http://www.weedscience.org/In.asp.
12. WSSA (Weed Science Society of America). Herbicide resistance and herbicide tolerance definition. Weed Technology. 1998; 12:789.