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Recent advances in herbicide resistance in weeds and its management

B. Duary
Institute of Agriculture, Visva-Bharati, Sriniketan (West Bengal)
E-mail: bduary@yahoo.co.in

ABSTRACT

Herbicides are the most effective and economic among the weed management practices. Use of herbicide is rapidly increasing in the world including India. Herbicides have revolutionized the weed management in world agriculture. Along with the advantages there are some inadvertent disadvantages like shift in weed flora, herbicide resistance and environmental concern. Development of resistance against the herbicides in targeted species is the most prominent among them. Herbicide resistance is a worldwide phenomenon and number of resistant biotypes of weeds is increasing at an alarming rate. Recently, almost one dozen species have been reported to be resistant against Monsanto's very potent broad spectrum herbicide glyphosate which has now become a key issue for all stakeholders. Sometimes the use of the term herbicide resistance is misleading. Before calling it herbicide resistance, the factors for poor efficacy of herbicide should be sincerely evaluated. It is essential to properly understand the herbicide resistance, its development and mechanism to tackle the problem. In this paper an attempt has been made to review of up to date information on current status of herbicide resistance in the world – development of resistance, factors controlling the development of herbicide resistance in weeds, resistance mechanisms, integrated approach of herbicide resistance management and lastly basic research and facilities required for better understanding of herbicide resistance and its management.

Key words: Herbicide, Resistance, Mechanism of resistance, Biotypes, Resistant management, Integrated weed management

Dr. B. Duary

Dr. Buddhadeb Duary is at present Reader in Agronomy in the Department of ASEPAN, Institute of Agriculture, Visva-Bharati, Sriniketan, West Bengal. He completed B. Sc. (Ag.) Hons. and M.Sc. (Ag.) in Agronomy from Palli Siksha Bhavana (Institute of Agriculture), Visva-Bharati securing first class second and first class first respectively and Ph. D. in Agronomy (Weed Science) from Indian Agricultural Research Institute, New Delhi securing first class first. Then he joined Pulses and Oilseeds Research Station, Berhampore, WB as Assistant Agronomist in January, 1999. On December, 2001 he joined Visva-Bharati as Lecturer in Agronomy. He was promoted to Senior Lecturer in 2003 and Reader in Agronomy with effect from January, 2008. He has been serving as the Principal Investigator of All India Coordinated Research Programme on Weed Control (ICAR) 2002 onwards, Area Coordinator, National Invasive Weed Surveillane (NIWS) Programme. He published more than 70 publications as research paper in National and International journals, in the proceedings of National and international Seminars/symposium, etc.

The response of plants to the severities of the environment has occupied the attention of man long before the beginning of the science of biology. 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 order to feed the ever-increasing population researchers were always in the lookout of new technologies which will increase the food production manifolds and are economically viable at the same time. Agricultural pests cause considerable loss in quantity as well as quality of agricultural produce worldwide. Among different biotic factors in an agro-ecological situation weeds are the major component causing maximum damage to the crop. It has been an established fact that weeds reduce farm yields and farm income drastically. So the introduction of pesticides in agriculture helped the farmers to control some

of the noxious pests and thus reduced the yield loss caused by them at an affordable cost. Among all other weed control practices, herbicides alone are most effective and economically acceptable mean. Herbicides have revolutionized the weed management in world agriculture. In the past three decades, evolution of newer herbicide molecules provided wider choice for the farmers (Duary and Mukhopadhyay 2004).

But along with these advantages there are some inadvertent disadvantages like shift in weed flora, herbicide resistance and herbicide residues in food chain. Development of resistance against these herbicides in targeted species was the most prominent among them.

Important definitions related to herbicide resistance

Before going for further discussion some related terms should be explained for proper understanding of herbicide resistance.

Site of action : Refers to the biochemical site within the plant where or with which the herbicide directly interacts. Many of the well-known sites of action are enzymes or proteins essential to plant growth and development also, some herbicides are believed to act at multiple sites.

Metabolism : Refers to the biochemical processes within the plant that generally modify herbicides to less toxic compounds. Differential rate of metabolism between crops and weeds is a primary method of crop selectivity to herbicides. One metabolic process may affect several different families of herbicides.

Herbicide families : A group of herbicides that share a common chemical structure and have similar herbicidal activity. Two or more herbicide families may affect the same site of action and therefore express similar herbicidal activity and injury symptoms.

A biotype : is a group of plants within a species that has distinct genetic variation not common to the population as a whole.

Herbicide susceptibility : It is the lack of capacity to withstand herbicide treatment with recommended dose so that the plant is damaged by the herbicide (Ashton and Crafts 1981).

Herbicide tolerance : It is the ability of a species to survive and reproduce after herbicide treatment. It is the ability to compensate the damaging effect of herbicides with No physiological mechanisms involved (Menalled and Dyer 2006).

Herbicide resistance : Refers to the inherited ability of a weed or crop biotype to survive a herbicide application to which the original population was susceptible. Thus, herbicide resistance is simply an altered response to a herbicide by a species which was earlier susceptible and it is the naturally occurring, irreversible and inheritable ability of some weed biotypes within a population (Duary and Yaduraju 1999).

Herbicides cross resistance : When resistsnce to two or more herbicidedes (with same or different mode of action) resulting from the presence of single resistance mechanism (one genetic mutation) 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 reesistance 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.

Multiple resistance: Multiple resistance is the phenomenon of resistance to herbicides from more than one chemical classes to which a population has been exposed (Holt *et al.* 1993). 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. For example, after a weed or crop biotype developed resistance to herbicide A, then herbicide B was used and resistance evolved to herbicide B. The plant is now resistant to both the herbicides A and B through two separate selection processes (more than one mutation). Multiple resistance was 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.

The term cross resistance should be used to describe cases in which a weed population is resistant to two or more herbicides by the presence of a single resistance mechanism. In contrast multiple resistance should be used in case where resistant plants possess two or more distinct resistance mechanisms.

One example of cross-resistance 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) also became resistant to several other ACCase inhibiting herbicides (Powles and Holtum 1990). On the contrary, a multiple resistance is said to have occurred when resistance to several groups of herbicides with different biochemical target such as triazines acting on PS system as photosynthetic electron transport inhibitor and sulfonylurea inhibit ALS i.e.

Acetolactate synthase enzyme (Menalled and Dyer 2006). In Southern Australia three herbicides, diclofopmethyl, chlorsulfuron and triasulfuron, had been used against annual ryegrass *Lolium rigidum* for almost 10 years. The diclofop-methyl resistant biotype revealed multiple cross-resistance to other groups (Powles and Holtum 1990).

Negative cross resistance: It refers to the phenomenon by which an individual resistant to one herbicide or a chemical family of herbicides shows higher or increased sensitivity or susceptibility to other herbicides than its natural wild type susceptible population. For example, the triazine resistant biotype of *Echinochloa crusgalli* which was 53 times more resistant than susceptible one, shows 33 and 2 times more sensitivity to fluazifop- butyl and sethoxydim, respectively (Gadamaski *et al.* 2000).

Current scenario of herbicide resistance in the world

Resistance of weeds to herbicides is not a unique phenomenon. In fact, resistance to pesticides is a world wide problem that is not confined to any single pest category. Although resistance of insects to chemicals was recognized as old as 100 years ago, the problem peaked up and was reported in several parts since 1940 after the use of synthetic organic pesticides was increased. Sanjos scales resistant to lime sulphur were sited in the year 1908. Later, pathogens resistant to fungicides were reported in 1940. Owing to the late commencement of use of herbicides in agriculture and probably due to the long generation cycle in plants, the resistance against the herbicide was the last to surface. Although herbicide resistance was reported as early as 1957 against 2, 4-D from Hawaii (Hilton 1957), the first confirmed report of herbicide resistance was against triazine herbicide in common groundsel (*Senecio vulgaris*) and was reported in 1968 from U.S.A. (Ryan 1970). Consequently, several other reports confirmed resistance developed against dozens of other herbicides in five decades (Table 1 and 2). The number of resistant weed biotypes against various herbicides is on the rise since its first report (Fig.1). Till July 10, 2008, 319 biotypes belonging to 185 species (111 dicots and 74 monocots) have been reported resistance against various herbicides [http://www.weedscience.org/Summary (July 10, 2008)] (Table 1 and 3). Though herbicide resistance in weeds is of recent origin, over the last 30 years, resistance in weeds has increased at a rate equivalent to that of insecticides and fungicide resistance (Heap 2007).

The problem of herbicide resistance initiated in 1960s was limited to only few species (<50) up to 1980s,but the numbe of resistant biotypes then increased exponentially to more than 250 in the next 20 years.

In recent years, the appearance of herbicide resistance in plants is increasing exponentially as compared with the case of other pests such as insects and fungi. Besides 101 species of Acetolactate synthase (ALS) or acetohydroxy acetone synthase (AHAS) inhibitors resistance, there are more than 200 species surfaced resistant to 18 other classes of herbicides. Herbicide resistance has been reported in 60 countries maximum in U.S.A. followed by Australia, Canada, France, Spain and U.K. etc. One third of total resistant weed biotypes were under triazine group fifteen years back. But for last 10 years the resistant weeds for ALS inhibitors have rapidly increased in number almost one and half time more than the number of triazine resistant weeds. About 95 weed biotypes are resistant to ALS inhibitors (till July 10, 2008) in 33 countries and are a great menace in cereals and soybean. One of the probable reasons is the widespread and larger use of the herbicide. For last 10 years ALS or AHAS inhibitors constitutes major share (17%) of total herbicide sale in the world. This group of herbicide is extensively used due to its high efficacy in lower dose, low mammalian toxicity, selectivity in more than twelve major crops (Das 2008). At least one weed species has emerged resistant to herbicide in each country of the world where herbicide is used as farm input. Recently, in 2003-2006, Monsanto's very potent broad spectrum herbicide glyphosate has been reported to be resistant against some weeds such as grassy weed Johnson grass, *Sorghum halepense* in Argentina (Valverde and Gressel 2006) and Rigid rye grass bio-type *Lolium rigidum* have exhibited resistance in almond orchard of northern California (Vargas 2001) (Table 2). Both hairy fleabane *Conyza bonariensis* and buckhorn plantain *Plantago lanceolata* have been reported glyphosate resistant in South Africa. Hairy fleabane has been difficult to control with glyphosate in California production system indicating possible resistance. Similarly, reports of poor or ineffective control of *Chenopodium* sp. in Roundup Ready cotton system have surfaced in the last few years. Recently, Roundup resistance horseweed *Conyza canadensis* has been confirmed in the eastern US. (Vargas and Wright 2004). This rapid widespread has been due to the fast airborne property of its seeds i.e. the seeds can travel 1/4th mile per mild wind speed of 10 MPH (Barnes *et al*. 2003). Over the past several years, the list of glyphosate resistant weeds grows to almost one dozen species (Table 1). Farmers are being challenged to control glyphosate resistant weeds like Palmer amaranth (*Amaranthus palmeri*) and giant ragweed (*Ambrosia trifida*) in certain crops. Monsanto commercialized glyphosate as RR (Roundup-Ready) with their respective genetically modified (GM) transgenic crop seed. This RR- seed package in soybean, corn, canola and cotton where offered a weed

Table 1. Herbicide resistant weeds as on July 10, 2008

Herbicide Group	Mode of Action	Example of Herbicide	Total number of biotypes
ALS inhibitors	Inhibition of acetolactate synthase ALS (acetohydroxyacid synthase AHAS)	Chlorsulfuron	95
Photosystem II inhibitors	Inhibition of photosynthesis at photosystem II	Atrazine	67
ACCase inhibitors	Inhibition of acetyl CoA carboxylase (ACCase)	Diclofopmethyl	35
Synthetic Auxins	Synthetic auxins (action like indoleacetic acid)	2,4-D	26
Bipyridiliums	Photosystem-I-electron diversion	Paraquat	24
Ureas and amides	Inhibition of photosynthesis at photosystem II	Chlorotoluron	21
Glycines	Inhibition of EPSP synthase	Glyphosate	14
Dinitroanilines and others	Microtubule assembly inhibition	Trifluralin	10
Thiocarbamates and others	Inhibition of lipid synthesis - not ACCase inhibition	Triallate	8
Triazoles, ureas, isoxazolidiones	Bleaching: Inhibition of carotenoid biosynthesis (unknown target)	Amitrole	4
PPO inhibitors	Inhibition of protoporphyrinogen oxidase (PPO)	Oxyfluorfen	3
Chloroacetamides and others	Inhibition of cell division (Inhibition of very long chain fatty acids)	Butachlor	3
Carotenoid biosynthesis inhibitors	Bleaching: Inhibition of carotenoid biosynthesis at the phytoene desaturase step (PDS)	Flurtamone	2
Arylaminopropionic acids	Unknown	Flampropmethyl	2
Nitriles and others	Inhibition of photosynthesis at photosystem II	Bromoxynil	1
Mitosis inhibitors	Inhibition of mitosis / microtubule polymerization inhibitor	Propham	1
Cellulose inhibitors	Inhibition of cell wall (cellulose) synthesis	Dichlobenil	1
Unknown	Unknown	Difenzoguat	1
Organoarsenicals	Unknown	MSMA	1
Total Number of Unique Herbicide Resistant Biotypes			319

Modified from http://www.weedscience.org/Summary/MOA Summary.asp July 10, 2008

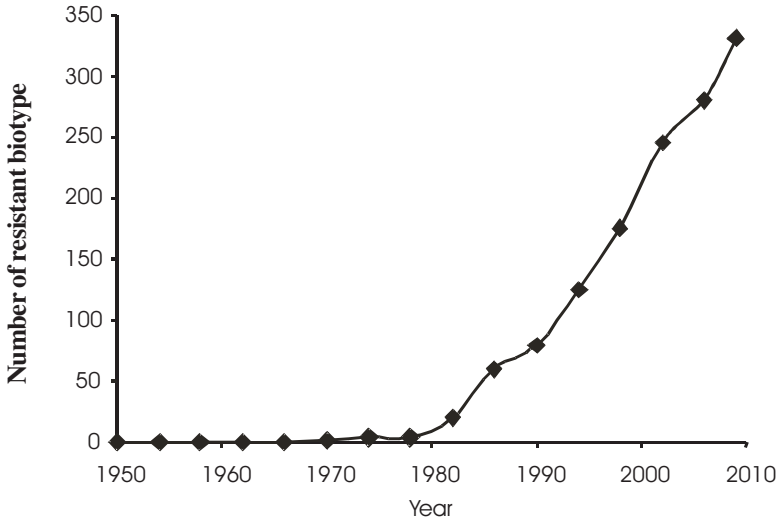

Figure 1. World wide chronological increase in the number of herbicide resistant weeds. [Modified from http://www.weedscience.org]

Table 2. Worldwide herbicide resistance against widely used herbicide groups

Herbicide	Year of resistance found	Year of reporting
2,4-D	1945	1963
Dalapon	1953	1962
Atrazine	1958	1988
Picloram	1963	1973
Trifluralin	1963	1982
Diclofop	1977	1982
Triallate	1962	1987
Chlorsufuron	1982	1987
Isoproturon	1992	1995
Glyphosate	2003	2006

(Modified from : Duary and Yaduraju, 1999, Valverde and Gressel, 2006)

Table 3. Most common genera of weed developing resistance worldwide

Common name	Genus	Number of documented occurrence of herbicide resistance
Pigweed	*Amaranthus*	42
Lambsquarters	*Chenopodium*	25
Fleabane/Horse weed	*Conyza*	22
Ryegrass	*Lolium*	21
Foxtail	*Setaria*	17
Wild oat	*Avena*	15
Barnyard grass	*Echinochloa*	15
Black grass	*Alopecurus*	13
Groundsel	*Senecio*	12
Knot weed/Smart weed	*Polygonum*	12
Night shade	*Solanum*	11

(Source : Vargas and Wright, 2004)

free high farm yield simultaneously, did evolve herbicide resistant biotypes, a challenge that has now become a key issue for all stakeholders. GM transgenic herbicide resistant crops are becoming volunteer weeds which are also associated with segregation and introgression of herbicide resistant traits in weed population that has ecological impact on plant communities (Owen and Zelaya 2004).

A consortium of expert committee: Herbicide Resistance Action Committee (HRAC), the North American Herbicide Resistance Action Committee (NAHRAC) and the Weed Science Society of America (WSSA) founded by Agrochemical Industry, have jointly focused to monitor the evolution of herbicide-resistant weeds and assess their impact throughout the world (Heap 2007). Global collaboration between weed scientists made the survey that claims dramatic number of weed species has developed resistance against variety of herbicides since 1980 (Fig.1).

The use of herbicide is still quite low in India as compared to developed countries. Rice-wheat cropping system is an important one in Indian food security system and is followed in about 11 million hectare. Littleseed canarygrass (*Phalaris minor*) a grassy weed, morphologically similar to wheat plants in its vegetative phase and very competitive, has established itself as number one pest in wheat from sixties particularly where rice-wheat system is followed contineous use of same herbicides isoproturon to control *P.minor* in wheat in rice-wheat system has resulted in development of resistant biotypes in some parts of north-west India. (Malik and Singh 1993, Yaduraju and Singh 1997).

Development of resistance

Continuous and repeated use of a herbicide or herbicides having same mechanism of action in intensive agriculture or horticultural system involving crop monoculture and minimum tillage have been the major causes of occurrence of herbicide resistance.

From the definition of herbicide it is clear that when any herbicide fails to produce its effect on a species it does not mean that resistance has developed. The efficacy of herbicide depends on many factors. Before calling it herbicide resistance the factors for poor efficacy of herbicide should be sincerely evaluated. Sometimes the use of the term herbicide resistance is misleading. It does not always mean that herbicide resistance will surely be developed if it is continuously used in a same area for long 1period. No case of herbicide resistance was reported from any of the centres of AICRP-WC like UAS, Bangalore, Visva-Bharati, Sriniktan, GBPAU &T, Pantnagar and 19 other centres where same herbicide has been used during last 10-12 years in long term permanent trial. This confirms that herbicide *per se* does not cause any mutation. Rather resistant gene is present in any of the single individual naturally in a large population over a large area. As evident from the definition of resistance, it is not due to the mutation caused by the herbicide as chemical, rather resistance appears from the selection of natural mutation that exist as small fraction of population of resistant plants. Herbicide-resistant plant biotypes are believed to be emerging from only one or a few plants that are already present in a population. It may be a single plant in a population of several millions. Although they look morphologically identical, minor invisible genetic differences do exist among them that confer inherent resistance against herbicides. Such a minute number of resistant plants continue to grow and expand by generation over time and seasons. When we apply a herbicide continuously for consecutive seasons, the susceptible plants of a weed decrease drastically and those resistant biotypes increase gradually to the extent

that we find that the herbicide appears to be ineffective at one point. At this stage we say that the weed has developed resistance against a herbicide or in other words called selection pressure of herbicides reached to maximum (Duke *et al*. 1991).

Confirmation of resistance

Before assuming and designating any weeds surviving herbicide application are resistant it is very much essential first to confirm whether the poor efficacy of herbicide is due to resistance or some other factors**.** In case a herbicide treatment fails to control weeds at a situation, weed resistance may not necessarily be the cause. Before assuming that any weeds surviving a herbicide application are resistant, one should rule out other factors that might have affected herbicide performance. Several factors would be faulty application, spurious materials, unfavorable weather conditions, improper timing of herbicide application and weed flushes after application of a non-residual herbicide. Before signing a positive statement for weed resistance to herbicide, the following parameters should be tested (Gunsolus 2008, Menalled and Dyer 2007).

(i) Herbicide label should be read carefully. Whether weeds listed on the product label are controlled satisfactorily or not. Chances are only one weed species will show herbicide resistance in any given field situation. Therefore, if several normally susceptible weed species are present, reconsider factors other than herbicide resistance as the cause of the lack of weed control.

(ii) Observe, if the uncontrolled weeds exist in patches and each patch contains different species, this case is not of herbicide resistance because it is very unlikely that all species will develop resistance. There could be one of several other reasons.

(iii) Check if the herbicide is used repeatedly at the same field and is of the same one mode of action. If "yes"chances are that weed is likely evolving resistant biotype.

(iv) Further, survey the area for any previous case of resistant weed reported. Interview growers for resistant-suspect to same herbicide. Did the same herbicide or herbicide with the same site of action fail in the same area of the field in the previous year?

(v) Also, investigate if the level of weed control on suspected weed was declining in the past few years.

If the answer to some of the above questions is "yes", chances are that the weed species in question is leading to herbicide resistance.

If the above diagnostic survey support that a certain biotype has likely evolved herbicide resistance, adequate sample of seed/plant material may be collected from the suspect population for the subsequent confirmation test by standard methodology step by step by bio-assay, plant assay/seed collection, greenhouse / plant-pot assay, dose response experiments, single dose resistance assay and specific discrete tests (Gunsolus 2008).

Factors controlling the development of herbicide resistance in weeds

Factors which are generally responsible for the development of herbicide resistance are disajesed to the selection pressure imposes resistance in plants. This selection pressure can be generated either by repeated use of one herbicide, or use of long residual soil applied pre-emergence herbicide or due to repeated application of same post emergence herbicide. Factors that stimulate the development of herbicide resistance are many folds; however, the key factors include weed characteristics, chemical properties of the herbicide and cultural practices (Vargas and Wright 2004) that are discussed below briefly.

Weed characteristics

The most likely weed characteristics that favour increased resistance against particular herbicides can be as following:

a) Initial frequency of the resistant individuals: The development of herbicide resistance in weeds is quite different from that of insecticide and other pesticide resistance. As discussed earlier, there is no case where herbicide induces a mutation. Resistant genotypes are present in natural plant population in varying frequency. Weeds with a diverse genetic background may have a resistant biotype that has a one in 1 million chance of occurring within a weed population. This is called initial frequency. Although these odds sound remote, a one weed in 1 million chance of occurrence can translate into a high probability of selecting for a herbicide resistant weed biotype unless proper methods to reduce selection intensity are used. The development of resistance on a field scale depends on the rate of increase in the proportion of the resistant genotype within population. If the initial frequency of the resistant individual is high in a natural weed population, then the resistance will surface more quickly than in a population where the frequency of the resistant individual is low, provided we are continuously applying the herbicide to which the biotypes exhibit resistance.

Repeated use of the same herbicide or herbicides having same mechanism of action, results in killing the susceptible biotypes allowing resistant individuals to

multiply and produce seed. Thus within a few season of application the whole population, is dominated by resistant biotypes. Dormancy and Gasquez (1990) found an average frequency of 3×10^{-3} triazine herbicide resistant individuals in a population of *Chenopodium album* and Powles *et al.* (1997) reported an average frequency of 1×10^{-2} diclofop-methyl-resistant individuals in a *Lolium rigidurm.* Though development of resistance depends on many other factors, other remaining the same, the resistance will be quicker in later case.

b) Selection pressure for evolution of resistant population: In a weed population when the herbicides are applied the susceptible individuals are killed and the pressure on resistant individuals to develop increases. More the killing of the susceptible individuals greater is the scope of resistant individuals to emerge. Thus, selection pressure is the relative proportions of resistant and susceptible individuals remaining after herbicide treatment (Gressel and Segel 1990). Application of herbicides having single target site and specific mode of action, longer soil residual effect, applied frequently and over several growing seasons without rotating, alternating or combining with other type of herbicides, impose a high selection pressure (Holt and LeBaron 1990). Again highly efficacious, pre-emergence herbicides also impose a strong selection pressure of resistance. A herbicide that control 99% of a susceptible population will leave considerably fewer susceptible individuals to contribute to the next generation than a herbicide that gives 80% control. This can make a noticeable difference in appearance of resistance (Duary and Yaduraju 1999, Das and Duary 1999).

c) Ecological/Biological fitness: Fitness measures the potential evolutionary success of a genotype. It may be defined as the reproductive success or the proportion of genes an individual leaves in the gene pool of a population (Warwick 1991). It is a single value of relative evolutionary success that combines both survival and reproduction. Fitness studies based on dormancy , germination, establishment, survival and biomass production revealed that triazine resistant plants are generally less fit than susceptible plants (Holt 1990). This is sometimes assumed to be the general phenomenon and an intrinsic feature of the herbicide resistance trait.

(d) Weed reproduction, seed production, seed dormancy and germination and seed bank in the soil: The seed bank in soil can exert a strong buffering influence in delaying the rate of appearance of resistance. For the species in which seed remains residual in the soil seed bank, the appearance of resistance will be delayed by the continued recruitment of susceptible individuals from the seed bank. Thus the importance of the buffer depends mainly on the germination dynamics and tillage or cultivation practices followed.

For a species if the seed residue is more in the soil seed bank, appearance of resistance will be delayed due to continuous recruitment of susceptible individual from soil seed bank. That is, nature will allow the resistant species to flourish only after major portion of the susceptible weed seeds have been exhausted from the soil. For this very reason the species that germinate readily from its propagules will develop resistance more quickly than those species whose propagules remain dormant in the soil. Because of hypersensitivity, with a single application the herbicide about 90-95% of the susceptible type is killed. So selection pressure will be high and resistance species evolve rapidly.

e) Nature of inheritance of resistant gene: Among other factors the mode of inheritance of resistance, gene flow, mode of pollination, levels of genetic variation to herbicide response, genetic exchange with susceptible population are important. Most cases of herbicide resistance are due to the action of a single gene with a high degree of dominance.

Herbicide characteristic

Continuous application of the same herbicide or different herbicide with the same mode of action will create selection pressure and will allow resistant population to flourish. The following properties of herbicide molecule build the resistance in weeds to label them as different biotypes.

(a) Herbicides with highly specific mode of action: If a herbicide has only one site of action in weeds, then a biotype need to be different in that particular site to be resistant. So the evolution of resistance against such herbicides will be quicker than against herbicides having multiple site of action.

(b) Herbicides metabolism: Herbicides that are subjected to enhanced metabolism in weeds have least chance to endure resistance in plants than weeds expressing resistance due to change at site of action. However , metabolism-based resistance to ACC-inhibiting herbicides is much less known although this type of resistance seems to be wide-spread (Delye 2005).

(c) Long residual activity : Long residual activity in soil will keep susceptible biotypes elim87inated or suppressed for longer time giving competition free growth autonomy to the resistant to flourish and reproduce.

(d) Over dependence on single herbicide : Frequent application of herbicides over large areas and several

seasons without any rotation, combination or alternative use with other herbicides having different mode of action facilitates the evolution of resistant biotypes of weed.

(e) Type of herbicide and its time and dose of application: Pre-emergence herbicides exert more selection pressure as these control weeds more efficiently right from the germination when compared with post emergence herbicides. Higher dose imposes more selection pressure and quicker development of herbicide resistance. Optimum dose should be applied if satisfactory weed control is achieved.

Cropping practice

(a) Tillage: Zero tillage favours the evolution of herbicide resistance as compared to conventional tillage as the susceptible individuals will be killed and number of resistance weed seeds in the surface layer will increase. There will be no renewal of susceptible weed seeds from deeper layer or no burial of resistance seeds to deeper layer.

(b) Cropping system: Monoculture favours same kind of weed and also force to use similar herbicide and cultural practices thus repeated use of same herbicide year after year leading to quicker evolution of resistance biotype. On the other hand crop rotation also facilitates herbicide rotation.

Resistance mechanisms

Several authors have reviewed the mechanisms of herbicide resistance in different herbicide classes. Dekker and Duke (1995) broadly grouped mechanisms of herbicide in to the following two categories:

Exclusionary resistance

Those that exclude the herbicide molecule from the site in plants where they induce toxic response. In exclusionary resistance mechanism the herbicide is excluded from the site of action in many ways. Resistance is caused in plants due to inaccessibility of the molecule at its site of toxic action. In other words, it is the inability of herbicide molecule to concentrate in right lethal amount at point of action within weed plant. This provides weed a blessed escape from death and avail a sort of herbicide resistance. Such exclusion of herbicide from the site of action can be due to several reasons.

(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).

(c) Sequestration and compartmentation: Herbicides are sequestered in many locations before it reaches the site of action. e.g. some lipophilic herbicide may become immobilized by partitioning into lipid rich glands or oil bodies (Stegink and Vaughn 1988). 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- Santhakumar 2003).

(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 PSII 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.

Site of action of resistance

(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 (Eberlein *et al*. 1999). In resistant biotypes the herbicide binding site of action is modified due to genetic change and thus resistant biotypes remain unaffected. This inherited modification to the herbicides sites of action is involved in the mechanisms responsible for most of the triazines, aceto1actate synthase (ALS) inhibitors and dinitroaniline herbicide resistance.

This target site based resistance is usually associated with resistance involving altered binding of herbicide to their target protein. This results from a single nucleotide change (mutation) in the gene encoding the protein to which the herbicide normally binds. This changes the amino acid sequence of the protein and reduces or destroys the ability of the herbicide to interact with the protein.

Acetolactate synthase (ALS) or Acetohydroxy acetone synthase (AHAS) catalyses the first reaction in isoleucine, leucine and valine (branched chain amino acids) production. Resistance to ALS inhibitors such as sulfonyl ureas (Ray 1984) and triazolopyrimidine (Subramanian *et al*. 1990) is due to an altered site of action by alternation of the gene encoding of ALS accompanied by production of a form of ALS that is insensitive to inhibition (Chaleff and Ray 1984, Saari *et al*.1990).

Dinitroaniline herbicides inhibit the formation of microtubules and thereby block mitotic cell division in susceptible plants. An altered target site was found in a biotype of *E1eusine indica* which is highly resistant to dinitroanilines. Resistance in this biotype is conferred by an altered form of tubluin that results in microtubule insensitivity to the dinitroanilines (Vaughn and Vaughan 1990).

(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.

Herbicide resistance management

As the resistance is essentially irreversible it is virtually impossible to reintroduce susceptibility into that community to its pre resistance level. It is important to start managing herbicide resistant weeds in initial stage of detection otherwise it may have serious consequences to control weeds in a safe and efficient manner. The main principle of effective management of herbicide resistance in weeds is to reduce the selection pressure for evolution of resistance. Therefore, the main focus should be on modifying those factors and practices which are responsible for quicker evolution of resistance e.g. repeated and continuous use of same herbicide, monoculture, reduced cultivation, over reliance on herbicide etc. The strategies of resistance management are as follows:

Herbicide management

a) Stop use of herbicide to which resistant developed

b) Use of alternative herbicides: It is clear that herbicide resistant plants are benefited in evolutionary sense when growing in a field situation where that herbicide is used repeatedly. Immediately after detecting the herbicide resistance the use of that particular herbicide should be stopped. The widespread use of herbicide with same mechanism of action hastens the evolution of resistance. Use of alternative herbicides having different chemistry and mechanism of action is recommended as a short term measure provided cost effective alternative herbicides are available.

c) Herbicide 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 for resistance biotype and delay the rate of evolution as compared to individual herbicide used alone. Mixing two or more herbicides each effective against different weed flora and each with different mechanisms of action are quite helpful in reducing the chance of shift in weed flora and the problem of evolution of herbicide resistance.

As previously discussed the use of same herbicide or a different herbicide but similar mode of action in consecutive years increases evolution of resistance. Reverse action can be achieved by using different herbicides with different modes of action in the subsequent season(s); this process is called herbicide rotation. Use of the same herbicide or different herbicides with the same mode of action will exasperate the problem of resistant weeds. So adopt rotation of herbicides with different mode of action. Perhaps tank mix application of herbicides could prevent or delay resistance pressure.

Evolution of target-site resistance to both vulnerable and partner herbicide, though possible when mixtures are used, are much delayed. The following reasoning based on compound resistance has been used to support this supposition. If frequency of individual resistant to each component of a pesticide in a mixture is independent in susceptible species, then joint probability of evolution of co-resistance to both herbicide in one individual equals

the product of the probabilities of resistance for each partner. Thus if a weed has a natural mutation frequency of 10^{-5} for resistance to vulnerable herbicide and 10^{-10} to mixing partner having different target site and if genes for resistance are inherited independently of each other, then the joint probability of resistance to both the herbicide in an individual will be 10^{-15} which is very rare (Wrubel and Gressel, 1994).

d) 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 a low risk for the selection of herbicide resistant weeds. On the other hand weeds have readily evolved resistance to ALS inhibitors, triazine, bipyridyliums, phenylures, and Acase inhibitors.

Herbicides having low residual activity in soil, applied as post-emergence are ideal for reducing selection pressure for resistant biotypes. Repeated use of herbicide, higher application rate and use of same herbicide over different season should be avoided as far as possible. Limit the use of soil-persistent herbicides.

Indiscriminate use of herbicide like pre-emergent application of herbicide must be avoided wherever there is an option for selective post-emergent herbicide. Adoption of herbicide resistant crops can also help us in this respect.

e) Use of herbicides with short residual life : If we are using herbicides having long residual life then the selection pressure will be more. Therefore use herbicides having short residual life in recommended dose.If we are increasing the dose of herbicide the residual period will be high.

Preventive method to stop the spread

Any weed management strategy applied to minimize selection pressure for resistance will block the emergence of resistance.

Threshold density of weed : The use of herbicide can be minimized, if prediction of threshold infestation of weed, that is severe enough to warrant herbicide use, is possible. This will help to maintain a proportion of both susceptible and resistant biotypes in the population and thus help to delay the rate of evolution of resistance.

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 and use of different weed control measures, the substituted crop can effect good control of weed. Growing the same crop every season will invite same inputs including herbicide because of the same ecological culture. Crop rotation allows manipulation of planting time, spectrum of weed infestation, cultivation techniques, choice of herbicide with different mode of action, different stage and different way of application. Using combination of weed control strategies offers a chance to eradicate the resistant biotypes and reduces the chances of their establishment.

Thus crop rotation may reduce the overall usage of herbicides and extend the feasibilities of using wide range of herbicide and ultimately reduces the selection pressure. Crop rotation could be an effective tool for *P. minor* control. As the problem of resistance in the *P. minor* is evident mostly in rice-wheat system, there is a scope for tackling the problem by altering sequence of cropping. Inclusion of sugarcane, pigeonpea or maize in *kharif* and or berseem, sunflower, mustard, barley or oat in *rabi* reduces problem of *Phalaris* (Duary and Yaduraju 1999). But crop substitution does not impress farmers due to a variety of reasons. Crop rotation could serve as a useful component in the Integrated Weed Management (IWM) programme.

Tillage practices : Minimum or reduced tillage systems require increased use of herbicide to control annual grasses and perennial weeds which are predominant in reduced tillage condition. In Zero tillage condition the weed seeds remain close to soil surface and susceptible individuals are killed by herbicides and also the chance of dilution of resistance from buried seed is reduced. On the other hand deep tillage and inversion type (mould board) tillage reduce the requirement of herbicide and delay the build up of resistance by burying the weed seeds deep and bringing the buried susceptible seed up and thus selection pressure is reduced.

Other cultural practices :

- Selection of weed competitive crop cultivars,
- Use of clean seed and certified seed
- Stale seed bed technique
- Closer row spacing
- Timely sowing and rate of seeding
- Good crop husbandry
- Soil solarization

Integrated Weed Management

Awareness

Participatory approach

Monitoring of herbicide resistance if any: Integrated Weed Management (IWM) practices

Exclusive reliance on any single highly efficient weed management practices- chemical or non-chemical may fail

within a reasonable time period due to evolutionary forces searching the way to escape from control. Integrated weed management is a viable strategy as it is based on the principle of using a wide range of control methods in appropriate combinations. In most of the cases the resistant problem has been solved by using the alternative herbicides. But it is noticed that new herbicides have greater propensity for development of resistance. Besides herbicide discovery, having new unique mode of action with all the characteristics necessary for regulatory and commercial success, is becoming more difficult. IWM strategies are the only solution in case of multiple resistance as it is the most complicated and in the worst cases - virtually no selective herbicide remains effective. Thus IWM strategies involving physical, chemical and biological in a integrated fashion without excessive reliance on any single method can help in successfully managing herbicide resistance while maintaining farm profitability and sustainability. Inclusion of all possible non-chemical weed control methods help more effectively against the weed resistance evolution. In contrast to no-tillage, cultivation practice stirs the soil, buries the early emerged weed seedlings (both susceptible as well as the resistant one) and solairizes the soil. Hand weeding eliminates the weed plants before the seed set, discarding a biotype, 90-100 percent. Mulching for organic matter will simultaneously debris the weeds before seeding, offering reduced weed population with crop stand.

In order to maintain check over the herbicide resistant biotypes, integrated weed management approaches as discussed above must be incorporated as appropriate. Crop rotation or preferably fallow tillage followed by close cultivation will keep the resistant population down. Extensive manual weed control by effective tools will offer 100 percent eradication of the suspected resistant biotype ensuring less emergence in the subsequent season. Ensure clean and certified seed is planted each season and clean farm machinery is driven in the farm. By the use of these varied weed control practices, farmers have (unconsciously) acted to avoid or greatly delay the emergence of herbicide resistant weed biotypes.

Basic research needs

Herbicide resistance in weeds is not new to science. Systematic studies on weed biology, weed ecology', genetics and mode of inheritance of resistance, pollination behavior, nature of gene flow, biochemistry and physiology of resistance are urgently needed for better understanding of causes and development of herbicide resistance in weeds. Research facility must extend window option for DNA finger printing test for resistant and susceptible biotypes. Investigation for genetic resistance (one gene or additive gene effect) must be available.

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 a diverse genetic background may 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. Through the employment of herbicides to control weeds in cultivated fields, we were moving against Nature' s laws of biodiversity. The Nature retorted with herbicide resistant weeds. But our battle against the pest is not inevitably the one we are going to loose, it must be fought as a complex war with all available weapons. Yet there is no reason to believe that we cannot maintain a satisfactory level of crop protection. System that involves the use of herbicides should always incorporate practices to prevent and manage for eventual occurrence of resistance. Over-reliance on herbicide should be minimized and herbicide should be used integrated with other practices. Herbicide should be used in rotation or as mixture. We must keep available all other alternative tools we ever had, including the manual, cultural and other practices which should be used in an integrated manner.

REFERENCES

Anderson RL and Barrett MR. 1995. Residual phytotoxicity of chlorsulfuron in two soils. *Journal of Environmental Quality* **14**: 517-21.

Ashton FM and Crafts AS. 1981. *Mode of Actions of Herbicides.* Second edition. John Wiley & Sons, New York, p. 525.

Barnes J, Johnson B and Glenn N. 2003. *Identify glyphosate resistant marestail/horseweed in the field.* Purdue University Extension.

Chaleff RS and Ray TB. 1984. Herbicide resistant mutants from plant cells. *Science* **223**: 1148-1151.

Chaudhry O. 2009. *Herbicide–Resistance and Weed-Resistance Management. Biology / Environmental Science* Albert Campbell Collegiate Institute (Con. Ed.) Toronto, Canada.

Das TK. 2008. *Weed Science – Basics and Applications,* Jain Brothers, pp.582-615.

Das TK and Duary B. 1999. Herbicide resistance in weeds: Current scenario, mechanisms and management strategies for now and future. *Annals of Agricultural Research* **20**(4):393-398.

Dekker J and Duke OS. 1995. Herbicide resistant field crop.*Advances in Agronomy* **54**: 69-116.

Delye C. 2005. Weed resistance to acetyl co-enzyme-A Carboxylase inhibitor: an update *Weed Science* **53** (5): 728-746.

Dormancy, H. and Gasquez, J. 1990. Appearance and spread of triazine resistance in common lambsquarters (*C. album*). *Weed Technol*ogy **4**: 173-77.

Duary B and Mukhopadhyay SK. 2004. Biotechnology and herbicides – the new tools to combat the weeds – the major cause of biotic stress in Agriculture. In *Volume of extended Abstract of Seminar on Biotic stress on Agro-ecosystems.* West Bengal Academy of Science and Technology Section of Agricultural and Forestry Sciences. Feb. 22, 2004. pp. 24-28.

Duary B and Yaduraju NT. 1999. Herbicide resistance in weeds – a potential threat to world Agriculture. *Pestology Annual* 1999, pp 16-27.

Duke SO, Christy AL, Hess FD and Holt JS. 1991.*Herbicide-resistant crops*. Comments from CAST 1991, Council of Agricultural Science and Technology, Ames, I.A.

Eberlin CV, Guittieri MJ, Berger PH, Fleming JK, Mallory-Smith CA, Thil DC, Baerg RJ and Belkmap WR. 1999. Physiological consequences of mutation for ALS-inhibitor resistance. *Weed Science* **47**: 383-392.

Gadamaski G, Carka D, Gressel J and Gawronski SW. 2000. Negative cross-resistance in triazine-resistant biotypes of *Echinochloa crusgalli* and *Conyza canadensis*. *Weed Science* **48**:176-180.

Gressel J and Segel AL. 1990. Modelling the effectiveness of herbicide rotation and mixtures as strategies to delay or preclude resistance. *Weed Technoolgy* **4** : 186-198.

Gunsolus JL. 2008. Herbicide Resistant Weeds. Online available in www.Herbicide Resistance.htm.

Heap IM. 2007. International survey of herbicide Resistance Weeds. Online internet available on http//www.weedscience.org/In asp.

Hilton HW. 1957. Herbicide tolerant strain of weeds. Hawain Sugar Planters Association *Annual Reports*. p.69.

Holt JS. 1990. Herbicide resistance. *Weed Technology* **4**:139-40.

Holt JS, Powles SB and Holtum JAM. 1993. Mechanisms and agronomic aspects of herbicide resistance. *Annual Review of Plant Physiology and Plant Molecular Biol*ogy **44**: 203-209.

Holt JS and Lebaron JM. 1990. Significance and distribution of herbicide resistance. *Weed Technology* **4**:141-149.

Malik RK and Singh S. 1993.Evolving strategies for herbicide use in wheat. Resistance and integrated weed management. In: *Proceeding of Int. Symp. Indian Society of Weed Sciencce*, Hisar, India,18-20 Nov, 1993-vol. I : pp 225-238.

Malik RK and Singh S. 1995. Littleseed canary grass (*Phalaris minor* Retz.) resistance to isoproturon in India. *Weed Technology* **9**: 419-425.

Menalled FD and Dyer WE. 2006. Herbicide-Resistance Weeds. In: *Weed Management Handbook* 2006-2007. Mantana, Uta, Wyoming. Editors: Dewey, S. Enole, S. menalled, F., Miller, S., Whitsides, R. and Johnson, L.

Menalled FD and Dyer WE. 2007. *Preventing and Managing Herbicide-Resistance in Montana*. MSU Exten. Mont. Guide. MT 200506AG.

Owen MDK and Zelaya I. 2004. Herbicide-resistant crops and weed resistant to herbicides. *Pest Management Science* **61**(3):301-311.

Ozair CA, Moshier LJ and Werner GM. 1987. Absorption, translocation, and metabolism of foliage-applied chloramben in velvetleaf (*Abutilion theophrasti*) and soybean (*Glycine max*). *Weed Science* **35**:757-762.

Powels SB and Holtum JAM. 1990. Herbicide resistance weeds in Australia. The Regional Institute Ltd. Farrer Centre. http:// www. regional.org.au/roc/1990/roc199001.htm.

Powels SB, Preston C, Bryan IB and Zutsum AR. 1997. Herbicide resistance: Impact and Management. *Advances in Agronomy* **58** : 57-93.

Ray TB. 1984. Site of action of chlorsulfuron. Inhibition of valine and isoleucine biosynthesis in plants. *Plant Physiology* **75** : 827-31.

Ryan GF. 1970. Resistance of common groundsel to simazine and atrazine. *Weed Science* **18**:614-616.

Saari LL, Cotterman JC and Primiani MM. 1990. Mechanism of sulfonylurea herbicide resistance in the broad leaf weed, *Kochia scoparia. Plant Physiol*ogy **93** : 55-61.

Stegink SJ and Vaughn KC. 1988. Norflurazon SAN-9789 reduced abscisic acid levels in cotton seedlings: A glandless isoline is more sensitive than it's glanded counterpart. *Pesticides Biochemistry Physiol*ogy **31**: 269-275.

Subramanian MV, Hung HY, Dias JM, Miner JH and Butler JH. 1990. Properties of mutant acetolactate synthase resistant to triazolopyrimidine sulfonanilide. *Plant Physiol*ogy **94**:239-44.

Tharayil-Santhakumar N. 2003. Mechanism of Herbicide Resistance in Weeds. On line book "Mechanism of Herbicides Resistance in Weeds". Weed Science Society of America. http:// www.weedscience.org/In.asp . Dated 13/8/2009.

Valverde B and Gressel J. 2006. Dealing with evolution and spread of *Sorghum halepense* Glyphosate resistance in Argentina. A Consultancy Report to SENASA.

Vargas RN. 2001. *Herbicide resistance*. Cooperative Extension Bulletin. UC Davis. USA.

Vargas RN and Wright SD. 2004. Herbicide resistance management. In : *Cotton Field Check*. Cooperative Extension Bulletin. UC Davis. USA.

Vaughn KC and Vaughan MA. 1990. Structural and biochemical characterization of dinitroaniline resistant *Eleusine Plant Physiol*ogy **62**:510-20.

Warwick SI. 1991. Herbicide resistance in weedy plants: Physiology and population biology. *Annual Review of Ecology Syst*em **22** : 95-114.

Wrubel RP and Gressel Z. 1994. Are herbicide mixtures useful for delaying the rapid evaluation of resistance? A case study. *Weed Technol*ogy **8** : 635- 648.

Yaduraju NT and Singh GB. 1997. Herbicide resistance in *Phalaris minor* –an emerging problem. *Indian Farming* **2** : 20-21&25.