HERBICIDE RESISTANCE IN WEEDS: A REVIEW

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ABSTRACT

At global level, herbicides constitute 47% of the total pesticides used during 1997-98. About 72% of the herbicide consumption took place in the developed countries viz., USA, Western Europe and Japan. In India, the herbicide consumption is 16% of the total pesticides used and it is expected to increase up to 20% by the end of the 9th five year plan. Though the herbicide consumption is very low in India, there are growing concerns and questions about herbicide residue in foodstuffs, soil, ground water and atmosphere, potential weed shift and resistance to herbicides. Research reports on the effect of continuous application of same herbicide for weed management indicate that weed resistance to particular herbicide. Herbicide resistant weeds develop because of selection pressure from the continuous use of chemicals with the same mode of action. Based on foreign work, we can know the chances for the occurrence of herbicide groups which are going to develop resistance in India.

DEFINITIONS

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 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 selection of variants produced by tissue culture or mutagenesis (WSSA, 1998).

Herbicide tolerance : is the inherent ability of a species to survive and reproduce after herbicide treatment. This implies that there was no selection or genetic manipulation to make the plant tolerant; it is naturally tolerant (WSSA, 1998).

Cross resistance : used to describes cases in which a weed population is resistant to two or more herbicides due to presence of a single resistant mechanism (Rubin, 1991).

Multiple resistance : used in cases where resistant plants possess two or more distinct resistant mechanism (Rubin, 1991).

OCCURRENCE OF HERBICIDE RESISTANCE

Ryan (1970) first reported the herbicide resistant weed, Senecio vulgaris in 1968 for triazine group herbicide (simazine), ornamental nursery in Olympia, Washington after 10 years of its continuous use. Le Baron (1992) reported that during 1990, 113 herbicide resistant weed biotypes had evolved in various locations worldwide. This included 58 species (41 dicotyledonous and 17 grass weeds) resistant to triazine herbicide such as atrazine. Triazine is the low to moderate risk of herbicide which developed resistance in 5 to > 10 years of its continuous application. 55 species (36 dicotyledonous and 19 grass weeds) resistance to other classes of herbicides. 84 species (59 dicots and 25 grass weeds) developed resistance to one or more herbicides. But during 1998, 234 herbicides resistant weed biotype have been recorded including Phalaris minor in India, in wheat fields (Singh, 1998). Development of herbicide resistance can be avoided by understanding the interacting factors involved in the development of resistance in a weed crop community (Gressel and Segel, 1990).

RESISTANCE TO PHOTOSYSTEM II (PS II) INHIBITOR HERBICIDES

Triazines, phenylureas, benzonitriles, triazinone and uracil herbicides inhibit e' transport in photosystem II. Although structurally diverse, these herbicides inhibit e' transport at common site (Bowyer et al., 1991). The following are the major weeds that developed resistance for triazine herbicides (a) dicots : Abutilon theophrasti, Amaranthus spp., Bidens tripartita, Chenopodium spp., Physalis longifolia, Polygonum spp, Solanum nigrum, (b) grass weeds : Bromus tectorum, Digitaria sanguinalis, Echinochloa crus-galli, Poa annua and Setaria spp. in USA, Europe, Canada, Newzealand and Isreal (Le Baron, 1992).

Le Baron and Gressel (1982) found that development of target site PS II resistance in
Triazine resistance is almost always due to mutation in the chloroplast PSbA gene that results in a single serine 264 to glycine amino acid change in the D1 protein of PS II (Bandeen et al., 1982). Because the D1 protein is a chloroplast gene product, triazine resistance is maternally inherited (Souza Machado et al., 1978). Furest and Norman (1991) also found that in triazine resistant weeds, resistance is due to the altered site in chloroplast. Weed Abutilon theophrasti developed resistance to atrazine via enhanced metabolism mediated by glutathione-S-transferase (Grownwald et al., 1989). Anderson and Grownwald (1987) studied resistance in Abutilon theophrasti found that resistance is controlled by a single, partially dominant nuclear gene that is not cytoplasmically inherited. Kremer and Kropff (1998) found that final dry matter production and berry production of triazine resistant Solanum nigrum in maize crop were lower than those of susceptible biotype in Netherland.

Sardar and Mallory Smith (1997) reported that triazine resistant pigweed (Amaranthus spp.) and common lambsquarters (Chenopodium album) in peppermint fields of Oregon. Le Baron (1991) found that atrazine resistant common lambsquarters was shown to be cross-resistance to bromoxynil. Atrazine resistant velvet leaf biotypes was 10 fold more resistant to atrazine than a susceptible biotype. (Gray et al., 1995).

In India, Malik and Singh (1993) conducted preliminary research in Phalaris minor resistance against isoproturon in Hisar, Haryana. This growing menace was also highlighted by Bhan (1994). Application of isoproturon at 1kg ha⁻¹ (35 DAS) showed resistance against Phalaris minor biotypes (Anil Dixit and Bhan, 1995). Application of clodinafop at 50-60 g ha⁻¹, fenoxaprop 100-120 g ha⁻¹ or sulfoalan at 20-25 g ha⁻¹ at 4 weeks after sowing has been recommended to control isoproturon resistant Phalaris minor (Yaduraju, 1999). Upward activity of cytochrome P450 mono oxygenases enzyme detoxify the substituted urea herbicides like isoproturon in plant system (Burnet et al., 1993). Isoproturon resistant biotype have sown cross-resistant to diclofop-methyl both under controlled and environmental conditions (Yaduraju and Ahuja, 1995; Kirkwood et al., 1997).

Julio Mendez et al. (1994) reported resistance in slender foxtail (Alopecurus myosuroides) for chlorotoluron in winter cereal monoculture field of north eastern Spain. Leach et al. 1994 found that in Echinochloa colona, both specific and total acyl amidase activity increased with plant age upto 15 days (four-leaf stage) then decreased beyond 20 days to about 50% of the maximum at 30 days in both resistant and susceptible biotypes. Specific activity with propanil in the resistant biotype was about 80% of that obtained for rice compared to 25% in the susceptible biotype. The specific activity of the amidase was 3-fold higher in the resistant biotype than in susceptible. Carrol-Mallory Smith (1998) reported that resistance to bromoxynil and terbacil in common groundsel (Senecio vulgaris) collected from peppermint fields of Oregon, were filed was treated repeatedly for the past 10 years, often more than once during a growing season. This is the first reported case of a bromoxynil resistant weed selected under field conditions. The bromoxynil rate required to reduce growth by 50% was 10 times higher for the resistant biotype than the susceptible biotype.

RESISTANCE TO ACETOLACTATE SYNTHASE (ALS) OR (ACETOHYDROXY SYNTHASE) INHIBITOR HERBICIDES

The chemically dissimilar sulfonyl urea, imidazolinone and triazolopyrimidine herbicide classes share a common target site. They catalyse the first reaction in branched chain amino acid like leucine, isoleucine and valine production in a biosynthetic pathway (Stidham, 1991). Resistance to ALS inhibitors has most often appeared in weeds of cereal cropping. Recently resistance in weeds of soybean in United States (Schmitzer et al., 1993) and rice in California and Australia (Hill et al., 1994). The first weed to exhibit resistance to ALS inhibitor was Lolium rigidum in Australia. For
sulfonylurea herbicides, in North America, biotypes of prickly lettuce Lactuca serriola, Russian thistle (Salsola iberica), Kochia (Kochia scaparia), perennial ryegrass (Lolium perenne) and common chick weed Stellaria medin (Saari et al., 1992; Hall and Devine, 1990). Devine and Eberlin (1997) reported that since 1987, more than 27 weed species in the world have been identified with resistance to ALS inhibiting herbicides. Resistance to ALS inhibitors herbicides are due to (a) an insensitive target site (Saari et al, 1994) (b) considerable diversity in mutations within ALS (Guttieri at el., 1995) (c) Non-target site resistance due to enhanced herbicide metabolism mediated by cytochrome P450 dependent microsomal oxidases (Cotterman and Saari, 1992).

Christopher et al. (1992) observed resistance in annual ryegrass (Lolium rigidum) for ALS inhibitors may involve at least two mechanisms, increased metabolism of the herbicide and or a herbicide insensitive ALS. Schmitzer et al. (1993) strongly indicated that the mutation in one particular ALS gene affects the binding of each ALS inhibiting herbicide differently, and it is not possible to predict cross resistance to other herbicides acting on the same target enzyme. The mechanism of population selected for weed resistance or genetically engineered crop resistance to a sulfonylurea herbicides is mainly related to insensitivity at the ALS target site due to differences in absorption, translocation (Saari et al, 1992). Danniel Anderson (1998) discovered the resistant shattercane biotype to primisulfuron under field conditions where the herbicide was treated for consecutively 3 years. Charlotte Eberlin et al. (1997) recorded 70, 40 and 9% higher concentrations of valine, leucine and isoleucine respectively on a per seed basis in sulfonyl urea Resistant Vs susceptible seeds of prickly lettuce (Lactuca serriola). Shaner (1991) reported that altered gene encoding for ALS enzyme was responsible for resistance in sulfonylurea and imidazolinone group of herbicides in weeds. Boutsalis and Powles (1995) found that the LD50 ratios of resistant Sonchus oleraceus for sulfonylurea and imidazolinone herbicides are greater than 64 fold and 4.5 fold respectively. All the resistant Sonchus oleraceus biotypes were susceptible to herbicides like MCPA, diuron and diflufenican, which do not inhibit ALS.

In South Goodwin Avenue, USA (Sprague et al., 1997) demonstrated that Amaranthus rudis (2 consecutive years treated with a mixture of chlorimuron and metribuzin) recorded > 1920 fold resistance at whole plant level and > 850 fold resistance at ALS enzyme level. Kassim Al-Khatib et al. (1998) found that resistance to imazethapyr was identified in a population of common wild sunflower that had been treated for 7 consecutive years. Resistant biotype was approximately 170 times greater resistance to imazethapyr than the susceptible based on the rate required for 25% control. Imazethapyr concentration required to inhibit invetro ALS activity by 25% was 210 fold higher in the resistant biotype.

Hall et al. (1998) opined that in Barley, false cleavers (Galium spurium) biotype was highly resistant to ALS inhibitors like triasulfuron, thifensulfuron/tribenuron, sulfometuron and moderately resistant to imazethapyr, where herbicides were applied 6 consecutive years. GR50 values were > 16, > 5, > 1.0 and > 9.9 respectively. The ALS enzyme activity (I50) values for triasulfuron, metsimeturon, chlorsulfuron, thifensulfuron and imazethapyr were 36, 34, 92, 96 and 14 times higher respectively for the resistant biotype compared to the susceptible biotype. Hinze and Owen (1997) found that common water hemp (Amaranthus rudis) population was cross resistance to both imidazolinone and primisulfuron. Dennis Tonks and Philip Westra (1997) reported that dicamba generally the most consistent and commercially acceptable herbicide for the sulfonylurea resistant kochia control over a range of conditions, biotypes and growth stages. However, dicamba use is restricted due to crop injury in wheat. Bromoxynil, bromoxynil + MCPA represent improved crop safety and have a broader application time, but must be applied to small kochia plants to ensure adequate control. Spurge et al. (1997) found that common cocklebur (Xanthium strumarium) was resistant to imazethapyr. GR50 for resistant biotype was > 390 than susceptible biotype. Common cocklebur was also resistant to imidazolinone, imazaquin. But also cross resistance to sulfonylurea, chlorimuron, and triazolopyrimidine. Mathew foes et al (1998)
reported that in *Amaranthus rudis* resistance to flumetsulam was due to substitution of leucine for tryptophan at residue 569 of ALS. This biotype was also cross resistant to thifensulfuron and flumetsulam.

**RESISTANCE TO ACETYL CO-ENZYME A CARBOXYLASE (ACCASE) INHIBITOR HERBICIDES**

This group comprises 2 major classes of herbicides aryloxy phenoxypropionate (AOPP) and cyclohexanediones (CHDS). Key products include flumazifop–butyl, fenoxaprop, sethoxydim, tralkoxydim and clethodim. These herbicides are only effective against grass weeds of broad leaved crops as well as in certain cereal crops in which there is selectivity. Accase are biotin containing enzymes found in plastids. Which catalyze ATP and HCO₃⁻ dependent conversion of Acetyl CoA to malonyl C–A. Malonyl Co-A is required for the synthesis and elongation of fatty acids and for synthesis of a number of secondary compounds (Walker et al., 1989). Several grass weeds have evolved resistance to ACCase inhibitors viz., *Alopecurus myosuroides* in Germany and United Kingdom, *Avena sterilis* and *Avena fatua* in Australia and North America, *Lolium multiflorum* in the United States, *Lolium rigidum* in Australia, Israel and the United States, *Setaria viridis* in North America (Jodie Holt, 1993). The existence of wild oat resistance to ACCase inhibitors, including both AOPP to CHDS was first confirmed in Manitoba, 1990 (Heap et al., 1993).

Many cases of ACCase herbicides resistance are due to modification of the target site (Devine and Shimabukuro, 1994). Enhanced metabolism (detoxification mechanism) endowing resistance has been observed for diclofop, fenoxaprop, or tralkoxydim in *Alopecurus myosuroides*, *Lolium rigidum* and *Digitaria sanguinalis* (Hall et al., 1993). Studies by Heap and Knight (1986) suggest that resistance to diclofop-methyl has originated independently due to increased selection pressures from herbicide use. Since ACCase inhibitor resistance is based on target site alternations, is controlled by single gene and inherited as a dominant or semi-dominant trait (Parker et al., 1990). Inheritance of resistance in *Avena sterilis* and *Lolium multiflorum* is due to a single, partially dominant, nucleus encoded gene (Barr et al., 1992; Betts et al., 1992; Mansooji et al., 1992).

Considering the selective link between herbicide resistance and seed bank persistence traits in Italian ryegrass, the use of crop rotations to reduce a monocultures selective pressure for the development of herbicide resistance (Gressel, 1991). Marshall et al. (1994) reported that resistant *Eleusine indica* biotype which is very resistant to fluazifop, shows an intermediate level of resistance to sethoxydim and fenoxaprop, but only a very low level of resistance to clethodim. In *Lolium multiflorum*, inhibition by diclofop was non competitive in both cases, but changed from linear to the sensitive enzyme to S-hyperbolic-l-hyperbolic in the resistant enzyme (Evenson et al., 1994). Gheras et al. (1994) suggest that shifting cereals from winter to spring would greatly hamper the development of Italian ryegrass weed populations (herbicide resistant and susceptible alike). Christopher et al. (1995) found that seedlings of rat tail fescue (*Festuca myuros*) red fescue (*Festuca rupa*) are resistant to diclofop-P and fluzifop-p-butyl and seedlings of red fescue exhibits resistance to sethoxydim upto 1.7 kg ai ha⁻¹. Andrews et al. (1998) opined that by using AFLP (Amplified Fragment Length Polymorphism) study, there was a strong association between the *Avena fatua* genotypes clustering of samples, their herbicide resistant characteristics and their field of origin. Improved sanitation of tillage and harvesting equipment and the use of certified seed could limit resistant seed movement of *Avena fatua*.

Heap and Knight (1986) found that *Lolium rigidum* evolved resistance to the ACCase inhibitor diclofop and exhibited cross resistance to sulfonylurea and imidazolinone. The resistance appears to be a quantitative, polygenic trait. It has been suggested that resistance or cross resistance to diclofop in *Alopecurus myosuroides* may be due to enhanced activity of cytochrome P₄₅₀, catalyzed detoxification system similar to that in wheat crop (Kemp and Caseley, 1992). Leach et al. (1995) found that ACCase from the resistant biotype of *Eleusine indica* was resistant to fluazifop (I₅₀ > 500µm), but much less resistant to clethodim (I₅₀ = 6.6 µm). In wild oat biotypes difference in cross resistance for diclofop showed that the
existence of multiple mechanisms of resistance, multiple alleles of one or more resistant genes that code for the site of action of the herbicide or both (Seefeldt et al., 1996).

**RESISTANCE TO PHOTOSYSTEM I (PS I) INHIBITOR HERBICIDES**

Two bipyridyl herbicides that primarily affect photosystem I are paraquat and diquat. Resistance to the herbicide paraquat has been documented in 18 dicotyledonous and 7 monocotyledonous weed species in worldwide. Paraquat resistance generally appeared following either multiple application (5-10) of herbicide over a number of years (5-7) or single annual applications for 12 or more years (Preston, 1994).

Vaughn and Fuerest (1985) found that in *Conyza bonariensis* application of 4.8 kg ai ha$^{-1}$ of paraquat there was no mortality in the resistant population. In *Conyza bonariensis* paraquat resistant can be conferred by more than one mechanism. Both reduced translocation and sequestration of paraquat away from the site of action. There are indications that increased amounts of detoxifying enzymes such as superoxide dismutase could also confer resistance to paraquat. Tucker and Powles (1991) found that in *Hordeum glaucum* (barley grass) application of 1.6 kg ai ha$^{-1}$ for resistant population, the mortality rate was only 33%. But susceptible population was controlled at 100 g ai ha$^{-1}$ of paraquat. Alizadeh et al. (1998) first reported paraquat resistance in zero tilled cropping system in world wide, in South Australia. In South Australia the field was used for continuous cereal cropping (wheat, barley). From 1980-1994 Paraquat was applied at the rate of 0.375-1 kg ai ha$^{-1}$. Based on LD$_{50}$ values, the resistant *Hordeum glaucum* populations demonstrated $>250$ fold resistance to paraquat. Even for 3.2 kg ai ha$^{-1}$ of paraquat majority of *Hordeum glaucum* weeds were recovered after application. But paraquat resistant *Hordeum glaucum* were normally controlled by fluazifop-p-butyl (or) oxyfluorfen or glyphosate (Alizadeh et al., 1998).

Resistance to paraquat could be conferred by increased ability to detoxify toxic forms of oxygen produced by electron transfer from the paraquat radical. Resistant weeds posses a number of protective enzymes capable of detoxifying toxic oxygen forms and thereby protecting cells from oxidative damage (Furest and Vaughn, 1990). Both paraquat resistant *Lolium perenne* and *Conyza* spp. were reported to have increased levels of superoxide dismutase in crude leaf extracts isolated. However, studies with resistant *Conyza canadensis*, *Conyza philadelphicus* and *Hordeum glaucum* showed no increase in activity of oxygen protective enzymes (Jodie Holt, 1993). Resistance in *Conyza bonariensis* (Shaalties et al., 1988) and *Hordeum glaucum* (Islam and Powles, 1988) were conferred by a single dominant nuclear gene.

Paraquat resistant *Conyza canadensis* was less vigorous than the susceptible biotype in the absence of paraquat (Itsh and Matsunaka, 1990). In Australia, Tucker and Powles (1993) recorded slight reduction in competitiveness of resistant *Hordeum glaucum* than susceptible, whereas resistant and susceptible *Hordeum leporinum* were equally competitive.

**RESISTANCE TO MITOTIC INHIBITOR HERBICIDES**

Dinitroaniline herbicides injure plants by interfering with the normal function of microtubules during cell division (Appleby and Valverde 1989). Evolved resistance to dinitroaniline herbicide has been reported in *Amaranthus palmeri* (Palmer amaranth) and *Eleusine indica* in United States, *Setaria viridis* (green foxtail) in *Carada*, *Lolium rigidum* (rigid ryegrass) in Australia and *Alopecurus myosuroides* (black grass) in England (Jodie Holt, 1993). *Eleusine indica* has highly resistant and *Setaria viridis* and *Lolium rigidum* biotypes have moderately resistant. Palmer amaranth is the first dicot reported with resistance to mitotic disrupters. Repeated use of this herbicides for 15-20 years resulted in resistance in weeds (Morrison et al., 1991).

Todd Andrews and Morrison (1997) opined that trifluralin resistance can persist in green foxtail populations for at least seven consecutive years of its use. Hugh Beckie and Morrison (1993) reported that *Setaria viridis* resistant biotype was seven times more resistant to ethafluralin than susceptible biotype. Highly resistant *Eleusine indica* biotype contain higher amounts of tubulin and a higher molecular weight form of β-tubulin than susceptible biotype (Vaughn and Vaughn, 1991). Kirankumar and Vance Baird (1995) found that
there was no significant difference in the banding pattern between the resistant and susceptible biotype for either \( \alpha, \beta, \gamma \) tubulin gene families by RFLP. Growth and development of several resistant biotype of *Eleusine indica* are apparently not impaired by the mutation, although dryweight is lower in resistant than the susceptible plants (Valverde et al., 1988). Resistant plants are less competitive than susceptible plants and respond to competition by reduced reproductive output.

**RESISTANCE TO EPSPS (5-ENOLPYRUVYL-SHIKIMATE-3-PHOSPHATE SYNTHASE) INHIBITOR HERBICIDES**

Glyphosate is the world's most widely used herbicide, accounting for 11% of worldwide herbicide sales (Powles et al. 1997). Glyphosate has been used extensively throughout the world for over 20 years. It is a non-selective herbicide with no soil activity. Glyphosate kills plants by inhibiting the biosynthesis of aromatic amino acids (Amrhein et al., 1980), specifically by inhibiting EPSP synthase, a key enzyme in the shikimate pathway. No naturally occurring glyphosate resistant biotype has been identified due to resistant EPSPS. Though more than two decades of use, no cases of herbicide resistance from field use have been reported for glyphosate (Bradshaw et al., 1997).

Powles et al. (1998) found that *Lolium rigidum* (rigid ryegrass) evolved resistance to glyphosate in Australia after 15 years of its use. *Lolium rigidum* exhibited 7-10 fold resistance when compared to a susceptible population. Some of the *Lolium rigidum* biotypes were cross resistant to diclofop-methyl (about 2.5 fold). Similar levels of control of the resistant and susceptible population of *Lolium rigidum* was obtained by following application of amitrole, chlorosulfuron, fluazipof-p-butyl, paraquat, sethoxydim, or tralkoxydim.

**RESISTANCE TO AUXIN-ANALOG INHIBITOR HERBICIDES**

Auxin type herbicides have been used extensively worldwide since their commercialization in the late 1940's. Despite their long term use, the incidence of resistance to these herbicides is low. These chemicals (2,4-D and MCPA) were first selective herbicide used to control dicot in cereal crops. One of the earliest recorded cases of resistance was in 1957 when Whitehead and Switzer (1963) discovered isolated populations of *Dacus carota* along road sides of Ontario in North America which were no longer sensitive to 2,4-D'. In 1990, Heap and Morrison (1992) found that population of *Sinapsis arvensis* in Western Canada which were resistant to 2,4-D and/or MCPA after 20 years of use.

In Europe, three dicots *Stellaria media*, *Cirsium arvense* and *Papaver rhoeas* and one grass weed *Echinochloa crusgalli* have been detected as tolerant to 2,4-D and quinclorac respectively (Coupland, 1994). Recently, farmers in Newzealand have found that after 25 years of 2,4-D or MCPA application these were less effective in controlling *Cardus mutans* (nodding thistle) and *Ranunculus acris* (buttercup) (Harrington, 1987). In Newzealand, populations of *Cardus mutans* requiring 5-30 times more MCPA and 2,4-D than normal recommended dose and *Ranunculus acris* being tolerant, required 4-8 times the dose of MCPA than susceptible populations. Dose response experiments conducted in the growth chamber indicated that wild mustard was resistant to 2,4-D and MCPA and highly resistant to dicamba.

In 1980, about 20 years after 2,4-D use, rice farmers of northern province in the Philippines reported that the broad leaved weed *Sphenoclea zeylanica* showed a high degree of tolerance to 2,4-D (Sy and Mercado, 1983). About nine years later in Malaysia, the sedge *Fimbristylis miliacea* was observed to survive 2,4-D treatments in direct seeded rice fields, where 2,4-D was applied one to two times in a season since 1975 (Ho, 1992). *Fimbristylis miliacea* resistance to 2,4-D was confirmed at rates 16 times higher than the use rate. The resistant biotype was cross resistant to MCPA but not to propanil, paraquat or glufosinate (Watanabe et al. 1994).

**CONCLUSIONS**

India too, despite its very limited use of herbicides by the farmers, is reeling under a grave situation of herbicide resistance in *Phalaris minor* (little canary grass) weed for isoproturon in wheat crop. Continuous use of isoproturon, a herbicide rated very economical in terms of controlling a broad spectrum of weeds and cost incurred in its use ha\(^{-1}\) in wheat since last 12-15 years, has
resulted in the evolution of resistant biotypes of *Phalaris minor*. It has become a potential threat to the sustainability of the rice-wheat cropping system in north-western plains of India.

New and high risk herbicides like sulfonylurea and aryloxyphenoxy propionate groups may cause resistance in weeds by 3-5 years of their continuous use. So introduction of high risk herbicides like sulfonylurea and aryloxyphenoxy propionate for weed management should be considered in India. Continuous use of paraquat in plantations, orchards may cause resistance in weeds. Butachlor and 2,4-D in rice fields may cause weed flora shift within 7-10 years. Low-moderate risk herbicides like atrazine and pendimethalin may cause resistance in weeds for 10-15 years of their continuous use.

REFERENCES


