

## Current status of herbicide resistant weeds around the Globe

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### ABSTRACT

The incidence and wide spread of herbicide resistant weeds is a global problem. Over the past 65 years, repeated use of herbicides has resulted in the evolution of resistant weed species. The first resistant species to triazine was discovered in 1970 in the United States. Since then, a large number of weed species has evolved resistance to several classes of herbicide. Currently, there are 334 resistant biotypes, including 190 weed species (113 dicots and 77 monocots) in over 310, 000 fields around the world. Common resistant species are *Chenopodium album* and *Amaranthus retroflexus* resistant to triazine, *Phalaris minor* resistant to isoproturon, *P. minor* and *P. paradoxa* resistant to diclofop, *Echinochloa colona* resistant to propanil, *Echinochloa crusgalli* resistant to butachlor, *Eleusine indica* resistant to trifluralin, *Lolium rigidum* resistant to diclofop, *Lactuca serriola* resistant to metsulfuron, glyphosate resistance to *Eleusine indica*, *Conyza canadensis*, *Lolium rigidum*, and *Lolium multiflorum*. Multiple weed resistance to more than one class of herbicides with different modes of action has also been documented with many species. Currently there has been increased herbicide resistance to various weed species around the globe. Most common species are *Lolium rigidum*, *Avena fatua*, *Amaranthus retroflexus*, *Chenopodium album*, *Setaria viridis*, *Echinochloa crusgalli*, *Eleusine indica*, *Kochia scoparia*, *Conyza canadensis*, and *Amaranthus hybridus*.

**Key words:** Herbicides, inhibitor, phytosystem and resistance

Modern crop production systems are highly dependent upon agrochemicals. During the past 60 years, the agrochemical industry has produced a range of increasingly sophisticated herbicides that have been used by the producers globally. However, heavy reliance on chemical weed control with a limited number of active ingredients or/and with limited cultural practices resulted in a number of weed species developing resistance.

The development of herbicide resistance in weeds is an example of evolution in plant species as a consequence of environmental as well as cultural changes brought about by man. Herbicide resistance is a result of selection for traits that allow weed species to survive under specific management practices that result in mortality of species. The evolution of resistance under continuous use of herbicides may be considered as an example of recurrent selection. Under this recurrent selection pressure, there is a progressive shift, sometimes may be rapid, in an average fitness of populations of weed species exposed to herbicide treatments. A selection pressure for herbicide resistance is also related to the efficiency of the herbicide, the frequency of use, and the duration of effectiveness. This paper reports the

current status of herbicide resistant weeds, mechanisms of resistance, management strategies, and future outlook.

### History of resistant weed species

The incidence of weed resistance to simazine, a triazine herbicide, was first discovered in 1970's in the state of Washington, USA (Ryan, 1970). The majority of triazine resistant weeds were identified in corn production in North America and Europe or in orchards in Europe. Since the discovery of the triazine resistant weeds in North America, the increased numbers of species and their distribution have been noted globally (Bhowmik, 2000; Heap, 2009). Multiple resistances to more than one class of herbicides with different modes of action have been found in Australia, Europe, and in North America. Currently, weed resistance have been noted with the following classes of herbicides: triazine, sulfonylurea and imidazolinone, aryloxyphenoxypropionate and cyclohexanedione, bipyridinium, urea/amide, dinitroaniline, glyphosate and other class of herbicides. Currently, there are 334 resistant biotypes, including 1190 species around the world. The resistant weed species include 113 broadleaf species and 77 monocot species (Heap, 2009).

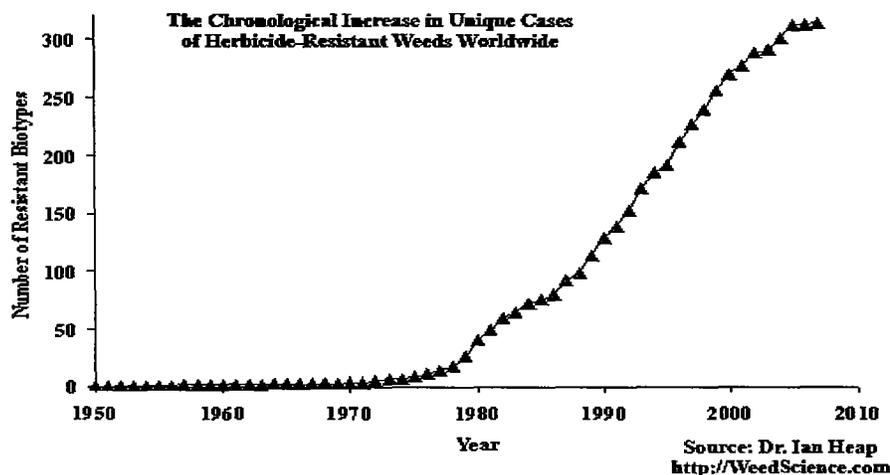


Fig. 1. The chronological increase in unique cases of herbicide-resistant weeds worldwide

#### Weed resistance

Herbicide resistance has been evolved through a period of time in our agricultural production systems. In this process, resistance mechanisms varied depending on the type of herbicides used under a weed management system for a specific crop. Mechanisms of resistance to herbicides is attributed to the following mechanisms: i) modified photosystem II protein-binding site (triazines and uracils), ii) modified ALS-binding site (sulfonylureas and imidazolinones), iii) enhanced detoxification (aryloxyphenoxypropionates and substituted ureas), iv) enhanced detoxification and /or sequestration (bipyridiniums), v) hyperstabilized microtubules (dinitroanilines), vi) EPSPS-related target-site alterations, vii) chloroacetamides and others.

The development of an herbicide-resistant weed population can be summarized by the following principle:

The appearance of herbicide-resistant weeds is the consequence of using an herbicide with a single site of action year after year or of repeating applications of an herbicide during the growing season to kill a specific weed species not controlled by any other herbicide or in any other manner.

This principle has three key components:

- An herbicide with a single site of action.
- Repeated use of the same herbicide.
- The absence of other control measures.

By understanding these components and developing weed-control systems with them in mind, growers or producers can greatly reduce the probability that herbicide resistant weeds will develop in their fields. There are two types of herbicide resistance identified around the globe as follows:

#### i) Cross-resistance:

Resistance to different herbicides with same mode of action or site of action. First, *Chenopodium album* was discovered as resistant to Atrazine. Later, the species was identified as resistant to simazine. Because these plants are resistant to both atrazine and simazine (Photosystem II inhibitors), they exhibit cross-resistance. Currently, more than 100 different herbicides are on the market today. But many of these work in exactly the same way or, in other words, have the same mode of action. Fewer than 20 plant-growth mechanisms are affected by current herbicides. ALS-herbicide resistance is a good example of the problem of cross resistance. ALS herbicides have been used for many crops. The Missouri-ALS-resistant *Xanthium strumarium* (common cocklebur) came from a field receiving only imazaquin (Scepter), but the cocklebur is cross resistant to primisulfuron (Beacon), flumetsulam (Broadstrike) and chlorimuron-methyl (Classic).

#### ii) Multiple-resistance:

Resistance to more than one class of herbicides with very different modes or sites of action in which more than one basis for resistance may be involved. A recent example of multiple-resistance is a biotype of *Amaranthus rudis* (common waterhemp) from western Illinois, USA. This biotype has demonstrated resistance to such herbicide families as the acetolactate synthase (ALS) inhibitors, triazines such as atrazine and simazine (photosynthetic inhibitors), and protoporphyrinogen oxidase (PPO) inhibitors.

In general, weed species having multiple resistance to herbicides are more difficult to control compared to weeds with cross resistance to herbicides. This is a key component to any management practices.

### General basis for weed resistance

What occurs within a resistant plant that allows it to survive after an herbicide application? What characteristics do the resistant plants possess that the susceptible plants lack? Two mechanisms have been identified that account for the majority of herbicide resistance cases.

#### i) Alterations in the target site of the herbicide.

A herbicide has a specific site within the plant where it acts to disrupt a particular plant processor function. If this target site is somewhat altered, the herbicide molecule may be unable to exert its phytotoxic action effectively. In general, most cases of herbicide resistance have involved alterations in the herbicide target site. Examples of this type include resistance to triazine (atrazine, simazine, and others), ALS-inhibiting herbicides (imazaquin, chlorsulfuron, and others), and ACCase-inhibiting herbicides (sethoxydim, fenoxaprop, and others).

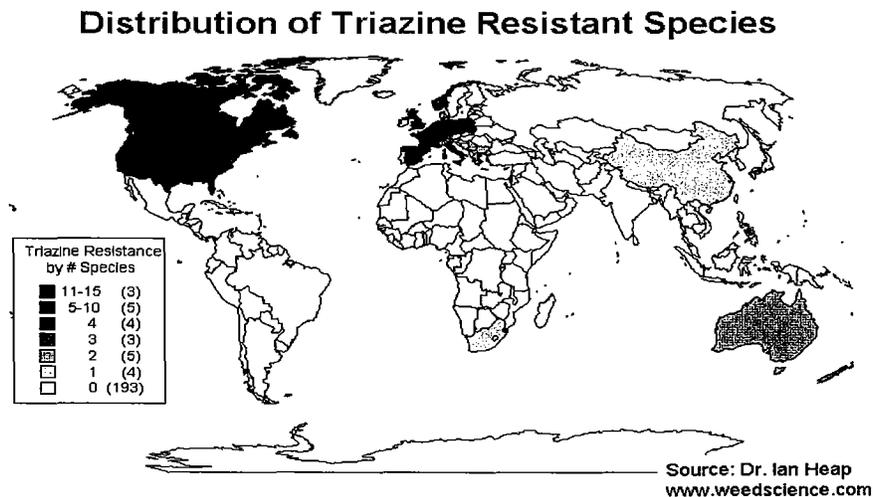
#### ii) Enhanced metabolism of the herbicide.

Metabolism within the plant is one mechanism a plant uses to detoxify a herbicide. A weed with an enhanced ability to metabolize a herbicide can potentially inactivate it before it can reach its site of action within the plant. A triazine-resistant biotype of *Abutilon theophrasti* (velvetleaf)

from Maryland, USA has been identified that possesses an enhanced ability to metabolize the herbicides, atrazine or simazine. Generally, as stated earlier, weed resistance to triazine herbicides is attributed to alterations in the target site of the herbicide. This velvetleaf biotype, however, possesses an enhanced enzyme activity that rapidly metabolizes the herbicide to nonphytotoxic forms. The mechanisms of resistance of various herbicides with different modes of action are explained briefly. These are not necessarily as complete as you will find with comprehensive review of the current literature.

#### Photosystem II inhibitors (C1/5)

The most frequently reported triazine resistant weed species are *Chenopodium album* (18 countries), *Amaranthus retroflexus* (13 countries), *Senecio vulgaris* (10 countries), *Solanum nigrum* (11 countries), *Amaranthus hybridus* (seven countries), *Amaranthus rudis* (two countries). Resistance of *Conyza canadensis* to triazine herbicides have been identified in nine countries. Other species such as *Solanum nigrum* (11 countries) and *Poa annua* (nine countries) have also been reported recently. The current distribution of triazine resistant weeds (68 species) is presented in Fig. 2.



**Fig. 2. World distribution of triazine resistant species**

In general, PS II inhibitors (triazines, phenylureas, pyridazines, and biscarbamates) compete for a common binding site on thylakoid membranes. Herbicide resistance can be due to two types of mechanisms: a) modified target site (altered site) and b) enhanced detoxification (altered metabolism).

Target site-based resistance is due to a modification of amino acid residues in the  $Q_B$ -binding niche on the D1 protein (Fuerst and Norman, 1991;

Trebst, 1991). This modification reduces the affinity of PS II herbicides at this site so that they no longer effectively compete for the exchangeable plastoquinone  $Q_B$ . In all cases of target site resistance that have occurred in the field, resistance is due to a point mutation of the psbA gene resulting in a substitution of Gly for Ser as residue 264 (Fuerst and Norman, 1991; Trebst, 1991). This mechanism of resistance is identified as altered site of action with

one gene inheritance at the chloroplast (Gronwald, 1994).

Resistance of *Abutilon theophrasti* L. to s-chloro-tiazines in the United States was due to an enhanced detoxification of atrazine via glutathione conjugation (Anderson and Gronwald, 1991). This mechanism of resistance is attributed to altered metabolism with one semi-dominant nuclear gene (Gronwald, 1994). Similar resistance was noted with *Lolium rigidum* to chlorotoluron in Australia (Burnet *et al.*, 1993). However, this resistance was due to enhanced detoxification via oxidative metabolism. Resistance to chlorotoluron can also be explained due to enhanced degradation by cytochrome p450 monooxygenase enzymes. This mechanism of resistance in *Alopecurus myosuroides* to chlorotoluron is due to altered metabolism with two nuclear genes (Moss, 1990).

#### ALS inhibitors (B/2)

ALS-inhibitors include sulfonylurea and imidazolinone herbicides. ALS-resistant weeds have been identified in cereals, corn, rice and other crops. *Lactuca serriola* L. (prickly lettuce) resistance to metsulfuron, a sulfonylurea herbicide, was first discovered in USA in 1987 (Mallory-Smith *et al.*, 1990), it is limited to two countries. *Kochia scoparia* (L.) Schrad. (kochia) is known to exhibit resistance to

ALS inhibitors and found in three countries (Sarri *et al.*, 1990). Several *Amaranthus palmeri*, *Xanthium strumarium*, and *Sorghum bicolor* have been evolved to exhibit resistance to ALS inhibitor herbicides. *Setaria glauca* resistance to imazapyr in the U. S. *Avena fatua* L. (wild oat) resistance to imazethabenz were found in four countries, but mostly in Canada. ALS resistant *Conyza canadensis* has been found in three countries and these exhibit multiple resistance. *Lolium rigidum* and *Lolium multiflorum* are also resistant to ALS inhibitors, and are primarily found in Australia and USA. Currently, there are 103 species resistant to ALS inhibitor type herbicides (Fig. 3).

Herbicides in this class inhibit the growth of plants via inhibition of acetolactate synthase (ALS), also known as acetohydroxyacid synthase (AHAS). The first target-site resistance to ALS inhibitor herbicide was identified in *Kochia scoparia* (Sarri *et al.*, 1990). *Stellaria media* (L.) Vill. (common chickweed), *S. iberica* and *Lolium perenne* L. (perennial ryegrass) were also identified as resistant to sulfonylurea herbicides (Sarri *et al.*, 1992). The target site resistance involves a decrease in the sensitivity of the herbicide target site to inhibition by the herbicide. This mechanism of resistance is attributed as altered site with one semi-dominant nuclear gene (Saari *et al.*, 1994).

### Distribution of ALS Resistant Species

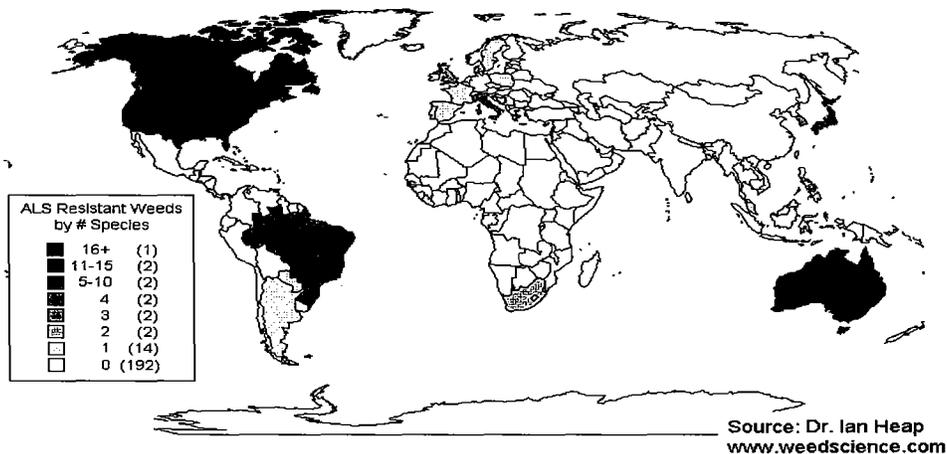


Fig. 3. World distribution of ALS resistant weed species ACCase inhibitors (A/1)

The ACCase inhibitors include Aryloxyphenoxypropionate and cyclohexanedione herbicides. There are now 38 species resistant to ACCase inhibitors (Fig. 4). The first occurrence of *Lolium rigidum* Gaud (rigid ryegrass) resistance to diclofop-methyl, ACCase inhibitor, was noted in Australia in 1982 (Heap and Knight, 1982), and this resistant species are found in 10 countries. Similar resistance to *Lolium multiflorum* have evolved to diclofop-methyl and other ACCase inhibitors in Chile,

France, South Africa, Spain, United Kingdom and the United States. *Avena spp.* has evolved resistance to ACCase inhibitors in 11 countries.

ACCase inhibitors include two sub-classes of herbicides. Aryloxyphenoxypropionate (APP) and cyclohexanedione (CHD) herbicides are used postemergence to control grass weeds in grass and broadleaf crops. The APP herbicides include diclofop-methyl, fenoxaprop-ethyl, fluzafop-butyl

and others, while CHD herbicides include clethodim, sethoxydim and others.

Mechanism of resistance is related to enhanced detoxification by resistant biotypes. ACCase forms in resistant species are less sensitive to these herbicides. It is possible that different ACCase

mutations may be involved (Marles *et al.*, 1993). This resistance of *Lolium rigidum* is due to altered site with one semi-dominant nuclear gene (Richter and Powles, 1993). Also, the resistance of *Avena fatua* L. to ACCase inhibitors is attributed to altered site with one semi-dominant nuclear gene (Murray *et al.*, 1995).

### Distribution of ACCase Resistant Species

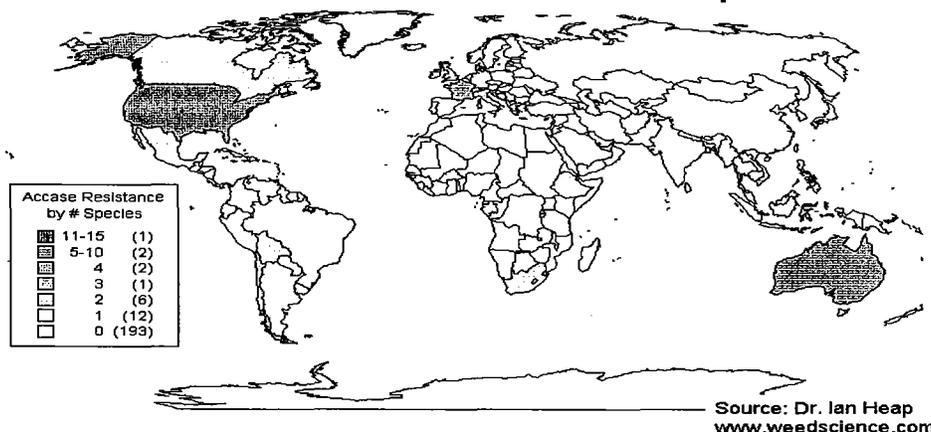


Fig. 4. World distribution of ACCase resistant weed species

*Lolium rigidum* selected with diclofop-methyl also results in multiple resistance mechanism, including the possibility of non-target site, metabolism-based resistance mechanism to ALS inhibitors (Christopher *et al.*, 1992; Powles and Howat, 1990). Mechanism of resistance to fenoxaprop-p-ethyl is characterized by the expression of both insensitive ACCase and an increased rate of detoxification of fenoxaprop-p-ethyl in *Alopecurus myosuroides* in United Kingdom (Cocker *et al.*, 1999), and now this resistant species is found in eight countries.

#### Glycine inhibitors (G/9)

In 1996, glyphosate resistant *Lolium rigidum* was identified in Northern Victoria, Australia (Pratley *et al.*, 1996) and one accession in South Africa (Heap, 2004), and now this resistant species are found in six

countries. *Lolium multiflorum* was identified as glyphosate resistant in California, USA (1998) and in Chilean orchards (Perez and Kogan, 2003), now is found in five countries. In 1997, glyphosate resistant *Eleusine indica* was discovered in Malaysia (Renolds *et al.*, 1999; Tran *et al.*, 1999; Lee and Ngim, 2000). *Conyza canadensis* (horseweed) in Delaware, U. S. was also identified as glyphosate resistant (VanGessel, 2001). Although this resistant *Conyza* species is common in the United States, it is found in three other countries. *Conyza bonariensis* (hairy fleabean) is also resistant to glyphosate and found in five countries. Glyphosate resistance to *Amaranthus palmeri* and *Amaranthus rudis* has been documented recently in the United States. Current world distribution of glyphosate resistant species (16) is presented in Fig. 5.

### Distribution of Glyphosate Resistant Species

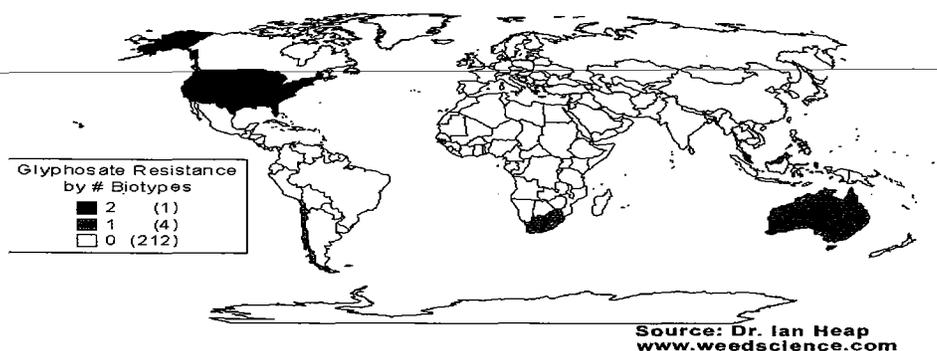


Fig. 5. World distribution of glyphosate resistant species

Glycine inhibitors are linked to EPSPS-related target-site alterations. The mode of action of glyphosate is based on inhibition of aromatic amino acids (phenylalanine, tyrosine, and tryptophane) biosynthesis which leads to arrest of protein production and prevention of secondary compound formation. Glyphosate inhibits 5-enolpyruvylshikimate-3-phosphate synthase (EPSP).

Earlier studies indicated that there were no significant differences in the uptake and translocation of  $^{14}\text{C}$ -glyphosate in the R and S biotypes of *Eleusine indica* in Malaysia (Tran *et al.*, 1999). The

biochemical data on sensitive and resistant biotypes of *Eleusine indica* showed a 5-fold difference in the  $I_{50}$  [glyphosate]s of the R and S 5-enolpyruvylshikimate-3-phosphate synthase (EPSPs). The RFLP analysis of the sensitive and resistant biotype DNA identified distinct molecular markers associated with the R biotype (Tran *et al.*, 1999). They also suggested that EPSPS-related sequences revealed distinct polymorphisms associated with the R biotype. The inheritance of resistance to glyphosate may be monogenic in nature.

**Table 1: Current status of glyphosate resistant weeds in the world.**

Weed species	Latin names	Year first reported	Location of resistant populations
<b>United States</b>			
Rigid ryegrass	<i>Lolium rigidum</i>	1998	California, other countries
Horseweed (maretail)	<i>Conyza canadensis</i>	2000	14 states
Italian ryegrass	<i>Lolium multiflorum</i>	2004	Oregon, other countries
Common ragweed	<i>Ambrosia artemisiifolia</i>	2004	Missouri, Arkansas
Palmer amaranth	<i>Amaranthus palmeri</i>	2005	Georgia, North Carolina, Tennessee
Common waterhemp	<i>Amaranthus rudis</i>	2005	Missouri
<b>World</b>			
Goosegrass	<i>Eleusine indica</i>	1997	Malaysia
Hairy fleabane	<i>Conyza bonariensis</i>	2003	South Africa, Spain
Broadleaf plantain	<i>Plantago major</i>	2003	South Africa
Johnsongrass	<i>Sorghum halepense</i>	2005	Argentina
Wild poinsettia		2005	Brazil

Source: *International Survey of Herbicide Resistant Weeds* ([www.weedscience.org](http://www.weedscience.org))

Currently, two major mechanisms of glyphosate resistance have been discovered in these two species: a change in the pattern of glyphosate translocation such that glyphosate accumulates in the leaf tips of resistant plants instead of in the shoot meristem, and amino acid substitutions at Pro 106 within the target site, 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) (Preston *et al.*, 2009). There are also populations with both mechanisms. In the case of glyphosate resistance, the target site mutations tend to provide a lower level of resistance than does the altered translocation mechanism. Each of these resistance mechanisms is inherited as a single gene trait that is largely dominant. As these ryegrass species are obligate outcrossers, this ensures resistance alleles can move in both pollen and seed. Some glyphosate-resistant rigid ryegrass populations appear to have a significant fitness penalty associated with the resistance allele.

*Multiple resistance to glyphosate in Lolium rigidum and Lolium multiflorum:* Where more than one mechanism of resistance to an herbicide occurs and individuals outcross, there is the potential for

multiple resistance to evolve. As used here, multiple resistance refers to the situation where an individual has resistance to one or more herbicides endowed by more than one resistance mechanism (Powles *et al.* 1997; Jasieniuk *et al.*, 2008; Nandula *et al.*, 2008). For glyphosate resistance in rigid ryegrass, there are at least two resistance mechanisms characterized; a target site mutation and reduced glyphosate translocation.

#### **Bipyridyliums (D/22)**

Paraquat resistance was first reported (1997) with *Poa annua* L (annual bluegrass) from hop gardens in the United Kingdom (Putwain, 1982), *Conyza bonariensis* (hairy fleabane) from vineyards and citrus plantations in Egypt (Fuerst *et al.*, 1985), *Eregeron canadensis* L. from vineyards (Kato and Okuda, 1983) and *Erigeron philadelphicus* (Philadelphia fleabane) in orchards in Japan (Watanabe *et al.*, 1982), and *Hordeum glaucum* in Australia (Bishop *et al.*, 1987). Similar resistance of *Conyza canadensis* (four countries), *Erigeron sumatrensis*, and *Youngia japonica* were also

observed. Currently, there are 24 paraquat resistant species documented in the world.

Paraquat and diquat are known as Photosystem I (PS I) disruptors. PS I is a membrane-bound protein complex which catalyzes the light-driven oxidation of plastocyanin (PC) and reduction of ferredoxin (Fd). PS I is made up of 10 to 11 proteins not including the chlorophyll a/b binding proteins.

Sequestration of paraquat as a mechanism for resistance has been proposed (Vaughn *et al.*, 1989; Fuerst and Vaughn, 1990). Increased binding of herbicide to cell walls is the mechanism for sequestration. Paraquat resistance can be summarized as the rapid sequestration followed by reduction of toxic level at the site of action in the chloroplast and a rapid enzymatic detoxification of superoxide and other toxic forms of oxygen. The mechanism of resistance of *Conyza bonariensis* is attributed to altered metabolism with one dominant nuclear gene (Darmency, 1994).

#### **Dinitroanilines and others (K1/3)**

Weed resistance to dinitroaniline class is limited to only 10 species in the world. *Eleusine indica* (goosegrass) resistant to trifluralin has been documented in 1982 and has continued to spread in the United States and Canada (Mudge *et al.*, 1984). *Sorghum halepense* (Johnsongrass) and *Amaranthus palmeri* (Palmer amaranth) resistance was also noted in the Southern United States. *Setaria viridis* (green foxtail) has evolved resistance to trifluralin in cereals and oilseed crops in North Dakota, USA and on the Canadian prairies (Morrison *et al.*, 1989).

The dinitroaniline herbicides, known as mitotic disruptors (hyperstabilized microtubules), include trifluralin, oryzalin, pendimethalin, prodiamine and others. Although the exact molecular mechanism for the inhibition is not known, it is believed that the dinitroanilines react with free tubulin heterodimers (composed of alpha and beta subunits) in the cytoplasm and, when attached to the growing end of the microtubule, prevent further polymerization (Vaughn and Vaughn, 1990). Resistance to *Eleusine indica*, *Setaria viridis* (Mudge *et al.*, 1984; Morrison *et al.*, 1989) and *Amaranthus palmeri* L. (Gossett *et al.*, 1992) has been noted with dinitroaniline herbicides. Resistance may be related to the differences in change in tubulin protein. The resistance mechanism in *Eleusine indica* is attributed to altered site with multi-nuclear genes (Smeda and Vaughn, 1994).

#### **Ureas/amides (C2/7)**

*Alopecurus myosuroides* (blackgrass/slender foxtail) resistance to chlorotoluron, a phenylurea herbicide, was reported in the United Kingdom in 1981 (Moss and Cussans, 1985). Resistance to phenylureas and amides has evolved in 21 species

around the world. Resistance of *Phalaris minor* (littleseed canarygrass) to isoproturon, a substituted area herbicide, has been noted in India in 1991 (Malik and Singh, 1995). In 2006, *Phalaris minor* resistant to isoproturon has been documented to exhibit multiple resistance to three MOA's. *Eichonochloa crusgallii* resistant to this class of herbicides have been reported in 1986, and now it is found in seven countries.

#### **Synthetic auxins (O/4)**

Resistance to 2,4-D was first identified in *Daucus carota* L. (wild carrot) in Canada (Switzer, 1957), now it is found in the United States. Other species such as *Papaver rhoeas* in Spain, *Synapsis arvensis* in Canada, and *Matricaria performis* in France were also known to be resistant to synthetic auxins. In 1992, quinclorac, a synthetic auxin, resistance in *Echinochloa crusgalli* was identified in rice fields of Spain. Currently, 28 weed species have been listed as resistant to auxin type herbicides.

#### **Chloroacetamides and others (K3/15)**

Propanil resistant *Echinochloa crusgalli* (barnyardgrass) was first reported in Chile (1993), and currently this species is found in Thailand, Philippine and United States. Resistant *Lolium rigidum* and *Lolium multiflorum* are also been reported in the United States.

#### **Protoporphyrinogen oxidase (PPO) inhibitors (E/14)**

*Amaranthus rudis* was reported to be resistant to PPO inhibitors in 2001 in Kansas, Illinois and Missouri, USA. *Ambrosia artemisiifolia* (common ragweed) was also found to be resistant this class. *Ephorbia heterophylla* (wild poinsettia) was first reported as resistant to PPO inhibitors in Brazil and it also exhibit multiple resistance.

#### **Consequences of Resistance**

Glyphosate-resistant weed species have several negative effects on a farm. For example, *Conyza canadensis* is a common winter annual weed that is best controlled with burndown treatments before no-till corn or soybean. But burndown glyphosate treatments and applications in Roundup Ready soybean have selected glyphosate-resistant plants that now infest millions of acres from Delaware to Illinois, USA.

In no-till fields, glyphosate-resistant *Conyza canadensis* poses a serious control challenge, so growers have tank mixed additional herbicides with glyphosate. Cloransulam-methyl has been a popular tank mix partner, but a significant number of glyphosate-resistant *Conyza canadensis* populations have also developed resistance to cloransulam-methyl and other ALS inhibitors. That's despite the fact that cloransulam-methyl has a different mode of action (ALS inhibitor) than glyphosate (EPSPS inhibitor). Currently, *Conyza canadensis* is known to have multiple resistance.

Some no-till growers have returned to using spring tillage to control horseweed that has glyphosate resistance or multiple resistances. Glyphosate resistance in this one weed may erode the gains that conservation tillage has made over the past 30 years. And because horseweed seed is easily spread by wind, even no-till growers who have adopted resistance management practices may be affected by this resistant weed.

Equally troubling, glyphosate-resistant waterhemp was reported in Missouri in 2005. Waterhemp infests millions of Midwest corn and soybean acres, and most waterhemp populations are already resistant to ALS-inhibiting herbicides (imazamox, imazethapyr, and others). There are also waterhemp populations resistant to PPO herbicides (fomesafen, lactofen, and others) and triazine herbicides (metribuzin, atrazine, and others). One Illinois farm has *Amaranthus rudis* population with three-way resistance (ALS, PPO, and triazine). *Amaranthus rudis* with multiple herbicide resistance that includes glyphosate resistance will be a true challenge to manage with herbicides.

For some weed species, glyphosate resistance may not be as serious because a tank mix partner could control the glyphosate-resistant weed. Even so, adding a second herbicide will be less convenient, increase costs, increase the risk of crop injury, and may limit the window of application.

#### **Glyphosate resistance – a consequence of repeated glyphosate use**

Glyphosate is the most widely used herbicide in world agriculture, as it is a broad-spectrum herbicide to control a wide range of annual and perennial plant species (Bhowmik, 2000; Duke and Powles, 2008). Glyphosate inhibits EPSPS, a key enzyme in the shikimate pathway in plants. This inhibition leads to a reduction in products of the pathway and a buildup of shikimate, eventually resulting in death of the plant. Since its introduction in 1974, glyphosate has been widely used for the control of weeds prior to crop seeding, inter-row spraying, in orchards, for weed control in non-cropped areas. In recent years, glyphosate has been widely used as a broad-spectrum weed control within glyphosate-tolerant crops. A weed's potential for developing glyphosate resistance is primarily guided by three factors such as weed characteristics, frequency of glyphosate use, and rates of glyphosate used.

##### **i) Weed species characteristics**

It is likely that certain weed species have greater genetic diversity, so there is a greater risk that they will develop herbicide resistance. Weed species that have already developed resistance to other herbicides may have a greater probability of developing glyphosate resistance. Species that may be prone to glyphosate resistance based on resistance to

other herbicide modes of action include *Amaranthus palmeri*, *Amaranthus rudis*, *Chenopodium album*, *Ambrosia species*, *Kochia scoparia*, and *Lolium species*. Since the trait for glyphosate resistance can spread by pollen or seed, the spread of resistant populations will be faster for some weed species than others.

##### **ii) Frequency of glyphosate used.**

Increasing the frequency of glyphosate use (number of applications on a given field) increases the probability of selecting a herbicide-resistant plant. With continued glyphosate use, the number of resistant plants will continue to multiply and create a resistant population. Herbicides do not cause the mutations that can also result in resistance. An extremely rare genetic trait that allows a weed to survive glyphosate applications may exist in the natural population. It is more likely to be found in a population that has been exposed to frequent application of glyphosate.

##### **iii) Rates of glyphosate used**

This is not clear how rate affects glyphosate resistance development in weeds. Several known glyphosate resistant weeds require eight to 10 times more glyphosate to be controlled than the normal, sensitive biotypes. This level of resistance means that labeled glyphosate rates will not control these weeds, and that making applications at labeled rates probably will not prevent resistance.

#### **Management of resistant weeds**

Management of resistant weed species is a challenging task. The tactics needed for an effective program are dependent upon weed species, crops to be treated, rotation crops, cultural practices used, and environmental conditions. The goal of resistant weed management is to delay or prevent the selection of a specific herbicide resistant population by reducing the selection pressure from one herbicide or a mixture of herbicides. Achieving these goals requires an understanding of the evolution and dynamics of resistant weed populations. This could be achieved with a sound weed management program, especially by adding diversity to our weed management programs.

Everyone would prefer a simple solution to glyphosate-resistant weeds. Diverse practices provide additional benefits since many of these practices improve the overall level and consistency of weed control, add flexibility in scheduling applications, and reduce the risks of yield loss. Overall, we must manage the intensity of glyphosate use to reduce the potential for resistance. These weed management practices avoid the continuous and exclusive use of glyphosate and lessen the potential for developing glyphosate-resistant weeds:

'An effective weed management strategy integrates the use of herbicides mechanical, and

cultural control methods. This type of approach would reduce the selection pressure. The following recommendations can be adopted for managing herbicide resistant weeds:

- Choose the herbicide program based on historical weed density and threshold information.
- Avoid using a single herbicide.
- Use a combination of herbicides with different modes of action.
- Rotate crops in a given field.
- Rotate herbicides with different modes of action (especially rotate glyphosate with other mode of action of herbicides).
- Use mechanical weed control methods, including cultivation, mowing, and others.
- Discourage extended use of a single herbicide or herbicides with the same mode of action on the same field.
- Apply a residual herbicide before glyphosate or tank mix with other herbicides with glyphosate.
- Avoid making more than two glyphosate applications to a given field over a two year period.

The global incidence and spread of resistant weed species will continue as agricultural production practices evolve with new technology. Based on weed biology or ecology, it is apparent that each weed species takes its own course to escape herbicide management strategy. Therefore, the complex interaction of weed species, herbicides, and the environment, including agricultural practices makes resistance mechanisms difficult to predict. We will continue to observe more species evolved through our management practices in the future.

Understanding the factors influencing the evolution of glyphosate resistance will help delay resistance appearance. For *Lolium rigidum* and *Lolium multiflorum*, the main factors are intensive use of glyphosate and the lack of other weed management practices. Orchards, vineyards, and fallow situations are at high risk of glyphosate resistance evolution.

Are other crops at risk? The frequency of glyphosate use is a major concern in corn and soybeans. The same concern exists in other cropping systems where glyphosate is commonly used. Heavy reliance on glyphosate in Roundup Ready alfalfa, canola, or other Roundup Ready crops in the future will also increase the potential for glyphosate-resistant weeds in those systems.

To date, there are few reports of glyphosate-resistant *Lolium* spp. from glyphosate-tolerant crops. This will certainly change as cultivation of glyphosate-tolerant crops become more widespread. Changing management practices to include other

weed control tactics will help delay the evolution of glyphosate resistance in these species, whereas the use of glyphosate as the only control method will exacerbate glyphosate resistance. In addition, the apparent fitness penalty associated with glyphosate resistance could be exploited in the management of glyphosate-resistant ryegrass by the inclusion of additional management strategies, including controlling seed set of surviving plants.

The global widespread of weed resistance to different classes of herbicides, especially to glyphosate has clearly reached the stage where more concerted efforts in research and education is required. We need to understand ecological implications of these species in production agricultural system, especially with worldwide adoption of Roundup Ready crops.

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