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Resistance of Weeds to Herbicides

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1. Introduction

Herbicides are the most widely used group of pesticides worldwide. The widespread use of herbicides has allowed tremendous gains in agricultural productivity worldwide. Since the 1950's herbicides have progressively replaced mechanical weed control because herbicides are more cost effective (Gianessi & Reigner, 2007). In 2009 over 95% of all the major agronomic crops grown in the U. S. were treated with herbicides (USDA-NASS, 2009). Transgenic herbicide-resistant crops were commercially introduced in the U. S. in 1996 when glyphosate-resistant (Roundup Ready®) soybean was released. Use of the very broad-spectrum herbicide, glyphosate, provided outstanding weed control (Dill, 2005; Dill et al. 2008). The most recent data indicates that the percent of the total acres of each of the following crops planted with glyphosate-resistant cultivars is soybeans 91%, canola 91%, cotton 71%, and corn 68% (Brookes & Barfoot 2009). Herbicides are used on >90% of arable farmland in the U.S. and herbicide-resistant crops has been used widely since the mid-1990's. Herbicide resistance in weeds was first discovered in 1968 (Ryan 1970) and there are currently 347 confirmed weed biotypes worldwide (Heap 2010).

When discussing pest resistance, whether it is weeds, pathogens, or insects, it is important to define the resistance. Some of the basic differences in the definitions of pest resistance depend on the basic definitions. The most basic unit of biological classification is the species, defined as a group of individual organisms displaying common characteristics and having the ability to mate and produce fully viable progeny. A population is a group of organisms within a species that co-occur in time and space (Radosevich et al. 1997) and share a distinct range of genetic variation. A species is usually composed of several to many populations. A genotype is the sum of the genetic coding or the genome of an individual. A biotype may not be fully coincident with genotype, as an individual has many genes. Certain genes may be expressed or unexpressed and not pertain to the phenotype associated with the biotype. A biotype is a phenotype that consistently expresses or exhibits a specific trait or set of traits. Weed scientists tend to refer to a biotype as a group of individuals with distinctive biochemical or morphological traits (e.g. resistance to a specific herbicide mechanism of action; growth and morphological traits). A phenotype refers to the physiological and morphological profile of the expressed genes in an individual. A single genotype can produce different phenotypes in response to environmental conditions present. This fundamental property of organisms is known as phenotypic plasticity. The alteration of phenotype (morphological or biochemical) without change in either the coding sequence of a gene or the upstream promoter region is classified as epigenetic change (Rapp & Wendel 2005). There is some controversy over whether

epigenetic changes can be inherited. The enhanced expression of EPSP synthase gene in glyphosate-resistant Palmer amaranth may be such a change.

The Weed Science Society of America's (WSSA) (1998) published its approved definitions for terms as follows: "Herbicide resistance (HR) is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering or selection of variants produced by tissue culture or mutagenesis." In herbicide-resistant crops, the resistance trait allows the use of a herbicide that would otherwise injure or kill the crop. Herbicide tolerance: "Herbicide tolerance (HT) 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." In crops, herbicide-tolerance allows the use of herbicides that control weeds but do not injure the crop.

Whereas there are many individual herbicide products, the Herbicide Resistance Action Committee (HRAC) recognizes only 16 unique modes of action (Senseman et al. eds. 2007), excluding those that are unclassified. The mode of action of an herbicide is the way the chemical controls the weed, thus it characterizes the selection factor. From the beginning of large-scale herbicide use, there were concerns about the potential for herbicide resistance (Appleby 2005). Like bacteria, fungi, and arthropods, weed populations adapt to selection; the most susceptible individuals are eliminated by exposure, while the less susceptible reproduce and present a succeeding generation that is more difficult to control than the former. The first case of herbicide resistance was to the triazine herbicide, simazine, in 1968 (Ryan 1970). Since then over 347 resistant weed biotypes have been reported; virtually all major modes of action of herbicide have certain weeds that have developed resistance to them (Heap 2010). During the 1970s and 80s different agronomic crops tended to use different combinations of herbicides, because the crops tolerated different herbicide modes of action, and generally more than one mode of action was needed in each crop to control the several species of weeds that might infest them. Since glyphosate had such broad activity against weeds, it was often used alone. Initially the argument was advanced that glyphosate resistance was highly improbable (Bradshaw et al. 1997). Nevertheless, a resistant biotype of rigid ryegrass (*Lolium rigidum* L.) was confirmed in Australia in 1996 (Heap 2010). There are now 18 reported instances of weed species that are resistant to glyphosate; they are found on all agriculturally productive continents.
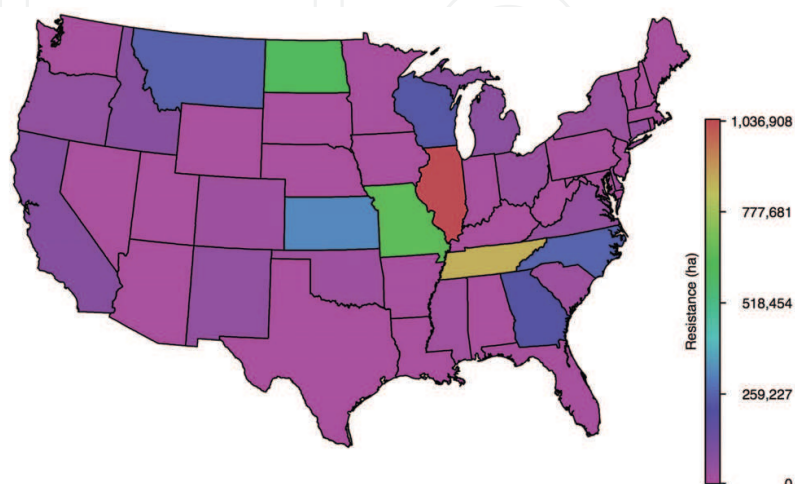

Fig. 1. Hectares of herbicide-resistant weeds in the US (Heap 2009).

Agronomic weed management is increasingly difficult and costly due to the apparent increase in the rate of development of weed resistance to herbicides and the lack of development of new modes of herbicide action. No new class of herbicides has been registered in the U.S. since mesotrione, an hydroxyphenyl pyruvate dioxygenase inhibitor in 1993. In contrast, the number of herbicide resistant weeds continues to increase, as have those specifically resistant to glyphosate.

Herbicide resistance in weeds occurs via target site resistance, enhanced metabolism, sequestration, reduced uptake, and over-production of the herbicide target site. Herbicide resistance has been confirmed to ten specific herbicide mechanisms of action. The most widespread resistance is to photosystem II-inhibitors, photosystem I inhibitors, acetolactate synthase (ALS)-inhibitors, acetyl-CoA carboxylase (ACCase) inhibitors, protoporphyrinogen oxidase (PPO or PROTOX) inhibitors, carotenoid synthesis inhibitors, EPSP synthase inhibitors (e.g. glyphosate), mitotic inhibitors, and auxinic herbicides. Within each of these herbicide mechanisms of action, there are multiple amino acid changes within the herbicide-binding domain. For many herbicide mechanism of action, there are multiple mechanisms of resistance possible. The specific mechanism can affect the level of cross-resistance observed. There are many factors such as herbicide rate can affect the type of resistance mechanism that occurs in the field. The presence of a fitness penalty associated with the resistance mechanism can also determine some dynamics of the herbicide resistance phenomenon.

The rapid adoption of herbicide-resistant crops has lead to a high dependence on a small range of herbicide mechanisms of action for weed management while suppressing the introduction of novel herbicide mechanisms of action. This increases the impact of weed resistance to one or two herbicide mechanisms of action can be economically devastating because of the paucity of alternative herbicide choices. Weed resistance to glyphosate in glyphosate-tolerant crops has become particularly problematic in areas of concentrated glyphosate-tolerant crop production. To minimize the spread of herbicide-resistance in weeds, growers will have to emphasize integrated weed management techniques of using cultural weed control, mechanical weed control, and using more than one herbicide mechanism of action to control targeted weed problems.

Of the weedy Amaranths, herbicide resistance has been reported in eleven species (Table 1). The first reported incidence of herbicide resistance in an agronomic crop in North America was in *Amaranthus hybridus* to the triazine herbicide atrazine in 1970 (Ryan 1970). *Amaranthus tuberculatus* biotype has been shown to have multiple resistance across three herbicide sites of action (ALS, PPO, PSII) (Patzoldt, et al., 2005).

Glyphosate- and ALS-resistant *Amaranthus palmeri* and *rudis* are of most concern and potential to disrupt current weed management systems in soybean, maize, and cotton in the United States.

2. PS II resistance

The first case of herbicide resistance in a row crop situation was *A. hybridus* to triazine herbicides in 1970 (Ryan 1970). Currently, triazine-resistant *Amaranthus* infests greater than 500,000 ha in North America. Resistance to photosystem II inhibitors is via target site resistance and enhanced metabolism. Target based resistance in the classical change in the Qb protein. The Qb protein is the site where electron transfer from chlorophyll to an initial electron acceptor, pheophytin, occurs in photosynthetic electron flow. Although many point mutations have been documented in cyanobacteria conferring resistance to triazine

Species	HRAC Group	Ha infested (worldwide)
Amaranthus albus	C1	250
Amaranthus blitoides	C1, B, C1 and B	4500
Amaranthus cruetus	C1	50
Amaranthus hybridus	C1, B	>75,000
Amaranthus lividus	B, C1, D	300
Amaranthus palmeri	B, C1, G, K1, B and G	1,000,000
Amaranthus quitensis	B	830000
Amaranthus retroflexus	B, C1, C2, B and C1, C1 and C2	>70000
Amaranthus rudis	B, C1, E, G, B and C1, B and E and G	>2,000,000
Amaranthus tuberculatus	B	250

Table 1. Herbicide Resistance in Amaranthus worldwide (Heap, 2010).

herbicides; in higher plants, only Ser-264-Gly, Val-219-Ile, and Ans-266-Thr have been documented (Patzoldt et al. 2003). However, the vast majority of triazine-resistance has been the Ser-264-Gly mutation. Other point mutations are less frequent. The Ser-264-Thr mutation confers resistance to triazine and substituted urea herbicides (Masabni & Zandstra, 1999).

In a survey of *A tuberculatus* in Illinois, 14 out of 59 randomly sampled populations were segregating for atrazine resistance, with only one of the 59 populations having site-of-action resistance (Patzoldt et al. 2003). The *A tuberculatus* population with site-of-action resistance, which was used in this study (UniR population), was also identified to have a second, non-site-of-action mediated mechanism. Thus, this novel triazine resistance mechanism may already be prevalent in *A tuberculatus* populations (Patzoldt et al. 2003). Similarly, an atrazine-resistant population of Amaranthus palmeri has been described in Georgia. This population seems to have enhanced glutathione conjugation of atrazine (Vencill 2008) and is not cross-resistant to other triazines such as ametryn.

The rate of CO_2 reduction in the *S*-triazine-resistant biotype of smooth pigweed (*Amaranthus hybridus* L.) was lower at all levels of irradiance than the rate of CO_2 reduction in the susceptible biotype. The intent of this study was to determine whether or not the lower rates of CO_2 reduction are a direct consequence of the same factors which confer triazine resistance. The quantum yield of CO_2 reduction was 23 ± 2% lower in the resistant biotype of pigweed and the resistant biotype of pigweed had about 25% fewer active photosystem II centers on both a chlorophyll and leaf area basis. This quantum inefficiency of the resistant biotype can be accounted for by a decrease in the equilibrium constant between the primary and secondary quinone acceptors of the photosystem II reaction centers that in turn would lead to a higher average level of reduced primary quinone acceptor in the resistant biotype. Thus, the photosystem II quantum inefficiency of the resistant biotype appears to be a direct consequence of those factors responsible for triazine resistance but a caveat to this conclusion is discussed. The effects of the quantum inefficiency of photosystem II on CO_2 reduction should be overcome at high light and therefore cannot account for the lower light-saturated rate of CO_2 reduction in the resistant biotype. Chloroplast lamellar membranes isolated from both triazine-resistant and triazine-susceptible pigweed support equivalent rates of whole chain electron transfer and these rates are sufficient to account for the rate of light-saturated CO_2 reduction. This observation shows that the slower transfer of electrons from the primary to the secondary quinone acceptor of photosystem II, a trait which is characteristic of the resistant biotype, is nevertheless still more rapid than subsequent reactions of photosynthetic CO_2 reduction. Thus, it appears that the lower rate of light-

saturated CO_2 reduction of the resistant biotype is not limited by electron transfer capacity and therefore is not a direct consequence of those factors that confer triazine resistance.

3. ALS resistance

Acetolactate synthase (ALS) is the first enzyme in the biosynthetic pathway leading to the synthesis of the branch-chain amino acids isoleucine, leucine, and valine. The branch-chain amino acids comprise part of the amino acid pool essential to protein synthesis and other plant functions. Inhibition of the ALS enzyme results in a cessation of growth followed by purpling of younger foliage then older foliage, as essential proteins cannot be synthesized. There are five chemical classes (sulfonylureas, imidazolinones, pyrimidinylthiobenzoates, triazolopyrimidines, and sulfonylaminocarbonyltriazolinones) that are confirmed to inhibit and ALS and these are used worldwide in numerous weed control situations in row crops and non-cropping situations. ALS resistance in is widespread in eight *Amaranthus* species (see Table 1). Of these, *A. hybridus* a *A. rudis* are the most widespread. There are documented cases of eight point mutations to the ALS gene conferring resistance to ALS-inhibiting herbicides. The Trp-574-Leu seems to be the most common and provides resistance to the greatest range of ALS inhibiting herbicides.

ALS-resistance in *A. rudis* had become so widespread in the midwestern US that ALS-inhibiting herbicides are not recommended (Syngenta press release). One of the reasons that glyphosate-resistant crops were adopted in the mid-1990's in the US so quickly and to such a great extent was because of ALS-resistance in the *Amaranthus* spp.

Point Mutation	Species	Resistance[a]
Ala-122 – Thr	*retroflexus, powelli*	IMI (SCT not tested)
Ala-205-Val	*retroflexus*	IMI (PTB, TP, SCT not tested)
Asp-376-Glu	*hybridus*	All groups
Pro-197-Leu	*retroflexus*	IMI, SU, PTB, TP (SCT not tested)
Pro-197-Ser	*blitoides*	PTB, SU, TP (SCT not tested)
Ser-653-Thr	*A.powelli, retroflexus, rudis*	IMI (PTB, TP, SCT not tested)
Ser-653-Asn	*rudis, hybridus*	IMI (PTB, SCT not tested)
Trp-574-Leu	*A.rudis, blitoides, retroflexus, powelli*	IMI, SU, PTB, TP, SCT

[a]IMI = imidazolinone, SU = sulfonylurea, PTB = pyrimidinylthiobenzoates,
TP = triazolopyrimidine, SCT = sulfonylaminocarbonyltriazolinone.

Table 2. Point mutations leading to ALS-resistance in Amaranthus spp. (Tranel et al. 2007)

There are no reported cases in Amaranthus where the ALS-resistance trait has lead to reductions in ecological fitness. *A. retroflexus* and *A. blitoides* were specifically examined and none were found.

4. PPO resistance

Amaranthus tuberculatus is only one of three species worldwide to develop resistant to PPO-inhibiting herbicides. Evaluation of a PPO-inhibitor-resistant *A. tuberculatus* biotype revealed that resistance was a (incompletely) dominant trait conferred by a single, nuclear gene. In plants, chlorophyll synthesis occurs exclusively in the plastids, while heme synthesis occurs in the plastids and mitochondria (Patzoldt et al. 2005). There are two nuclear genes to encode

PPO isozymes in the plastid and mitochondria. These are called PPX1 and PPX2 for the plsatid and mitochondria, respectively. Protogen IX accumulates in sensitive plants treated with PPO inhibitors. Protogen IX exported to the cytoplasm is converted to proto IX that in the presence of light causes the formation of singlet oxygen that results in membrane damage and eventual plant death. One gene from the resistant biotype, designated PPX2L, contained a codon deletion (G210) (Patzoldt et al 2005). PPX2L is predicted to encode both plastid- and mitochondria-targeted PPO isoforms, allowing a mutation in a single gene to confer resistance to two herbicide target sites. Resistant biotypes of *A. tuberculatus* have robust resistant to most PPO-inhibiting herbicides (lactofen, sulfentrazone, flumioxazin). Deletion of a codon rather than substitution is a unique formation of target site resistance to herbicides. There have been no studies to determine if there is a fitness costs to PPO resistance in weeds.

5. Glyphosate resistance

Glyphosate-resistance was first confirmed in *Lolium rigidum* in 1996 from Australia (Heap 2010). There are nineteen biotypes of weeds that have confirmed glyphosate-resistance worldwide. The most widespread resistance in from *Conyza canadensis*, first cofirmed in Delaware in 2001. It is estimated to infest more than three million hectares in the US alone. The first reported case of glyphosate-resistance in an in-season row crop was in *Amaranthus palmeri* in 2005. Currently, glyphosate-resistance has been confirmed in *A. palmeri, A. rudis*, and *A. tuberculatus*. Culpepper et al (2006) showed that the mechanism of resistance differs from that described in *Conyza candadensis* and *Lolium spp*. Glyphosate-resistance in a Amaranthus palmeri is due to increased EPSPS expression (Gaines et al 2010). While increased expression of EPSPS as a molecular glyphosate resistance mechanism has been reported to endow relatively low level glyphosate resistance in lab studies, this is the first report in a field weed population. It is likely that glyphosate selection pressure over several years in the Georgia cotton field (3) either selected plants with previously existing *EPSPS* gene amplification, or *EPSPS* gene amplification occurred during a period of less than seven years over which glyphosate was repeatedly applied. If we examine glyphosate-resistant *Amaranthus palmeri*, we see at least two mechanisms of resistance (reduced translocation and a target site change) and perhaps biotypes with both types of resistance as well as individuals that are resistant to glyphosate and ALS-inhibitors. Other collections of Palmer amaranth that seem to have very low levels (<2 x) of glyphosate resistance that have been difficult to characterize may be a third type of resistance. Before weed scientists can effectively manage glyphosate-resistant Palmer amaranth as well as other glyphosate-resistant weed species, we will need to better characterize at the genetic level whether individual plants are resistant via translocation mechanism, target site, combinations of these, and whether they are resistant to other herbicide mechanisms of action. Sammons et al. (2007) suggest that there are three primary mechanisms which confer herbicide selectivity among plants: 1) differences in herbicide target sites, 2) inactivation of an herbicide by chemical modification (i.e. metabolism), and 3) exclusion mechanisms which either reduce herbicide uptake or sequester the herbicide away from the target site. To clarify the exclusion mechanism, Ge et al. (2010) reports that glyphosate-resistant *Conyza canadensis* actively transports glyphosate to the vacuoles of the cell compared to the cytoplasm preventing it from getting to the target site.

Greenhouse data indicate that the glyphosate-resistant *A. palmeri* may have a fitness cost. The GS biotype grew at an 11% faster rate than the GR biotype, and the GR biotype assimilated carbon at 60.2% the rate that the GS biotype assimilated carbon. Measurements

of photosystem I activity, chlorophyll content, and branching help to characterize the GR biotype of Palmer amaranth, and suggest a mechanism of resistance different from that of *Conyza canadensis* and some other confirmed glyphosate-resistant weed biotypes, but did not correlate with relative fitness differences.

Glyphosate resistance has been particularly troublesome in the central U. S. including the states of Illinois, Missouri, Arkansas, and Tennessee. Glyphosate-resistant horseweed was first discovered in Delaware (van Gressel, 2001), but quickly spread to Indiana (Davis et al., 2007, Davis et al. 2008), Tennessee (Steckel & Gwathmey, 2009), and Arkansas. Glyphosate resistant horseweed increased the cost of weed management by about $13/acre (Mueller et al. 2005). While troublesome, glyphosate-resistant horseweed is primarily a problem at pre-plant before crop establishment. The emergence of glyphosate-resistant Palmer amaranth (*Amaranthus palmeri*) and water hemp (*Amaranthus rudis, A. tuberculatus*) have caused severe and well-documented management problems for in-season weed management in cotton and soybeans (Culpepper et al. 2008; Legleiter et al. 2008, Legleiter et al., 2009; Norsworthy et al., 2008a; Norsworthy et al. 2008b; Patzoldt et al. 2002; Patzoldt et al. 2005; Steckel & Sprague, 2004a; Steckel & Sprague, 2004b; Steckel et al. 2007; Steckel et al., 2008; Volenberg et al. 2007).

6. Mitotic inhibitor resistance

There are a number of herbicide classes that inhibit mitosis via disruption of microtubule formation. These include dinitroaniline herbicides such as trifluralin, pendimethalin, and ethalfluralin as well as some pyridine, carbamate, and phosphoroamidate herbicides. Microtubules are an integral part of mitosis as well as other cellular process such as cytokinesis and vesicular transport (Powles and Yu, 2010). These herbicides bind to one of the α- and β-tubulin dimers. Sensitive plants symptoms include malformed root areas that come in contact with the herbicide. Resistance occurs through a Thr-239_Ile substitution in the α-tubulin gene resulting in reduced binding of the herbicide. Resistance to mitotic inhibiting herbicides is not widespread with evolved resistance reported in 10 species worldwide (Heap, 2010). Resistance has been reported in South Carolina in *Amaranthus palmeri* in 1994 (Heap 2010) and a population was found with resistance in Georgia in 2010 (Vencill, personal communication).

7. HPPD-inhibitor resistance

Three classes of chemistry (triketones, isoxazoles, and callistemones) are bleaching herbicides that inhibit 4-hydroxyphenyl pyruvate dioxygenase (HPPD), a key enzyme required for the formation of carotenoids. The inhibition of carotenoid synthesis by the inhibition of the HPPD enzyme leads to white foliage because the carotenoid pigments protect chlorophyll pigment in plant tissues. Carotenoid synthesis can be inhibited by two other herbicide mechanisms of action, the inhibition of phytoene desaturase (e.g. norflurazon and fluridone) and the inhibition of deoxyxylulose 5-phosphate synthase (DXP) by clomazone. Resistance has been confirmed for all three bleaching herbicide mechanisms of action. Fluridone (phytoene desaturase inhibition) resistance is widespread in hydrilla in Florida and clomazone-resistant barnyard grass is reported in rice production in Arkansas and Louisiana. Resistance has been reported in a population of Amaranthus rudis in Illinois (Ag News, 19 July 2010). The mechanism of resistance is not understood, but resistance seems limited to foliar applications of HPPD-inhibiting herbicides while soil applications of the same herbicides seem to still provide control.

8. Muliple resistance

In the United States, the only documented case of resistance to multiple herbicide mechanisms of action has been in the Amaranthaceae. Cases of multiple resistant to ALS and PSII as well as ALS and glyphosate. Describe the ALS/PSII. In Georgia, there are biotypes resistant to ALS and glyphosate but little is known about specifics.

There are several populations of *A. tuberculatus* that have evolved multiple herbicide resistances. An Illinois biotype has resistance to PSII, ALS, and PPO inhibitors while a population from Missouri has evolved resistance to ALS, PPO, and EPSPS inhibitors (Patzoldt et al. 2003 According to Mueller (2005), there are >150000 ha of PSII/PPO/ALS-resistant common waterhemp in Illinois. In Georgia, populations of *Amaranthus palmeri* have been documented to be resistant to ALS and EPSP inhibitors. There are populations of A. palmeri that are reported to be resistant to mitotic inhbitors, ALS, and EPSP inhibitors.

9. Conclusion

In Europe, *Alopecurus* has been documented to a weed of serious agronomic potential to have evolved widespread resistance to commonly used herbicides and to multiple mechanisms of action in some cases (Delye 2005). In Australia, the niche is occupied by *Lolium* where resistance is documented to several groups of herbicides (Neve et al. 2004). In the United States, *Amaranthus* has long been one of the most common and troublsome weeds in agronomic crops and has been of the first weeds to develop resistance to herbicides in many situations. They were the first weeds to develop resistance to triazine herbicides, ALS-resistance in A. tubercualtus was widespread in the mid-1990's before the introduction of glyphosate-resistant crops, and glyphosate-resistance has been found in three species of *Amaranthus* and is growing rapidly. PPO-inhibiting herbicides have become the standard recommendation for glyphosate-resistant *Amaranthus* spp. However, we now see PPO-resistant *A. tuberculatus*. There are unconfirmed reports of resistance in A. palmeri in the southeastern US. The first case of multiple herbicide resistance in the US was in *Amaranthus tuberculatus* and *palmeri.*

In the past, herbicide resistance in *Amaranthus* caused growers to shift to another herbicide mechanism of action. There has only been one new herbicide mechanism of action introduced since 1990 so we are to a crisis point where growers may not have another herbicide mechanism of action to go to when resistance to PSII, ALS, PPO, and EPSPS inhibitors become more widespread in one of our most common and troublesome weed species. Without the introduction of new herbicide mechanisms of action or better herbicide-resistance management, a technology that has allowed tremendous increases in agricultural productivity is at risk.

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Herbicides and Environment

Edited by Dr Andreas Kortekamp

Herbicides are much more than just weed killers. They may exhibit beneficial or adverse effects on other organisms. Given their toxicological, environmental but also agricultural relevance, herbicides are an interesting field of activity not only for scientists working in the field of agriculture. It seems that the investigation of herbicide-induced effects on weeds, crop plants, ecosystems, microorganisms, and higher organism requires a multidisciplinary approach. Some important aspects regarding the multisided impacts of herbicides on the living world are highlighted in this book. I am sure that the readers will find a lot of helpful information, even if they are only slightly interested in the topic.

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