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Herbicide cross resistance in weeds

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ABSTRACT

With no major new site-of-action herbicide introduced into the marketplace in the last 20 years, the stagnation or decline in available herbicides in the past decade in a number of jurisdictions, and ever-increasing incidence of herbicide-resistant (HR) weeds, more efficient use of our existing herbicide tools will be required to proactively or reactively manage HR weed populations. Herbicide-resistant weed management can be aided by crop cultivars with alternative single or stacked herbicide-resistance traits, such as synthetic auxins, which will become increasingly available to growers in the future. An examination of cross-resistance patterns in HR weed populations may inform proactive or reactive HR weed management through better insights into the potential for HR trait-stacked crops to manage HR weed biotypes as well as identify possible effective alternative herbicide options for growers. Clethodim is the lowest resistance risk acetyl-CoA carboxylase (ACC) inhibiting herbicide, with only two of eleven target-site mutations (amino acid substitutions) in weed populations that confer resistance. However, there are no reduced-risk acetolactate synthase/acetohydroxyacid synthase (ALS/AHAS) herbicides or herbicide classes. Growers will be increasingly reliant on reduced-risk herbicide sites of action (groups), such as microtubule assembly inhibitors (e.g., trifluralin, pendimethalin), synthetic auxins (e.g., 2,4-D, dicamba), some photosystem-II inhibitors (nitriles such as bromoxynil), protoporphyrinogen oxidase (PPO) or hydroxyphenylpyruvate dioxygenase (HPPD) inhibitors, glyphosate, or glutamine synthetase inhibitor (glufosinate), used in sequences, mixtures, or rotations, to manage HR weed populations.

1. Introduction

No major new site-of-action herbicide has been introduced into the marketplace for about 20 years; the capture of a large fraction of the herbicide market by glyphosate with the commercialization of glyphosate-resistant crops beginning in 1996 contributed to significantly diminished herbicide discovery efforts worldwide (Duke, 2011a). Concomitantly, stricter pesticide registration requirements and environmental regulations in a number of jurisdictions have resulted in a drastic decline in available pesticides. This loss is greatest in Europe, with 945 active substances in 1999 compared with 336 in 2009, a 64% reduction (Moss, 2010). Among these compounds were some formerly very widely used herbicides, such as atrazine and trifluralin. Therefore, at least in the short term, more efficient use of our existing herbicide arsenal will be required to combat the ever-increasing incidence of herbicide-resistant (HR) weeds worldwide.

Proactive or reactive HR weed management can be aided by crop cultivars with alternative single or stacked herbicide-resistance traits, which will become increasingly available to growers in the future. Combinations of herbicide-resistance traits including glyphosate (group G; Anonymous, 2011), glufosinate (group H), acetolactate synthase/acetohydroxyacid synthase (ALS/AHAS) inhibitors (group B), hydroxyphenylpyruvate dioxygenase (HPPD) inhibitors (group F2), and synthetic auxins (2,4-D, dicamba; group O) can be stacked in crop cultivars (Feng et al., 2010; Green and Castle, 2010). For example, crops with stacked traits may include glyphosate plus dicamba-resistant soybean [*Glycine max* (L.) Merr.]; corn (*Zea mays* L.), cotton (*Gossypium hirsutum* L.), and soybean resistant to glyphosate plus 2,4-D plus acetyl-CoA carboxylase (ACC) inhibitor (group A); and glyphosate plus glufosinate-resistant corn (Reddy and Norsworthy, 2010).

This strategy is generally viewed as giving enhanced flexibility to growers to cost-effectively manage weed resistance through herbicide mixtures and sequences within a growing season, or herbicide rotations across growing seasons, provided that sufficient herbicide site-of-action diversity is maintained in rotations involving crops with stacked traits (Green et al., 2008; Carpenter and Gianessi, 2010; Culpepper et al., 2010; Owen, 2010). The latter caveat is critical to the

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sustainability of crops with stacked herbicide-resistance traits. Weed populations resistant to glufosinate or HPPD inhibitors were first reported in 2010 or 2011 (Jalaludin et al., 2010; Seng et al., 2010; Avila-Garcia and Mallory-Smith, 2011; Hausman et al., 2011; McMullan and Green, 2011). If crops with stacked herbicide-resistance traits are managed similarly as many of the current glyphosate-resistant crops, the same problems of weed shifts and evolved herbicide resistance will occur (Owen, 2010).

With the trend of stagnant or declining number of available herbicide sites of action in both major- and minor-area crops in the face of ever-increasing incidence of herbicide resistance in weeds, an examination of cross-resistance patterns in HR weed populations within and across sites of action (i.e., groups) may inform HR weed management. As described below, the cross-resistance pattern in an HR weed biotype depends upon the type of resistance mechanism. Better insights may be gained into the potential for future crop cultivars with alternative single or stacked herbicide-resistance traits, as listed above, to proactively or reactively manage HR weed biotypes based on knowledge of cross-resistance patterns. In addition, many growers want to know which herbicides are still effective in controlling HR weed populations in their fields. This information is best obtained by collecting suspected HR weed shoot tissue or seed samples for herbicide resistance testing (Beckie et al., 2000) to identify alternative or remaining herbicide options either within the same group as the herbicide suspected of selecting for resistance or from a different group. Knowledge of the prevalence of particular cross-resistance patterns within or across herbicide groups may aid this identification. Accordingly, this review provides a comprehensive global synopsis of herbicide cross-resistance patterns in weeds.

2. Cross-resistance patterns in weeds

By definition, cross resistance between two or more herbicides in an HR weed biotype is conferred by a single mechanism. A single gene contributes to a single mechanism, although it is possible for more than one gene to contribute to the same resistance mechanism (Preston and Mallory-Smith, 2001). The mechanism can be target site-based, i.e., mutation at the site of herbicidal action, or nontarget site-based, such as altered metabolism or translocation. Both enhanced metabolism and reduced translocation in HR biotypes prevents phytotoxic levels of herbicide from reaching the site of action. Enhanced metabolism is generally responsible for cross resistance across herbicide sites of action (i.e., groups), whereas cross resistance attributed to altered target site or translocation is usually restricted to herbicides with the same site of action.

Multiple resistance in an HR biotype is commonly defined by two or more mechanisms, and is usually the result of sequential herbicide site-of-action selection or accumulation of resistance alleles in progeny as a result of pollen flow in outcrossing species such as *Lolium rigidum* Gaudin, *Alopecurus myosuroides* Huds., *Kochia scoparia* (L.) Schrad., and a number of *Amaranthus* spp. Herbicides can select for any pre-existing mechanism conferring resistance in weed populations, which has been repeatedly demonstrated in the above-mentioned weed species. The incidence of intergroup herbicide resistance, due to cross resistance (i.e., metabolism-based mechanism) or multiple resistance, is continually increasing. To date, there are about 50 weed species with intergroup herbicide resistance (Fig. 1).

Lack of effective alternative herbicide sites of action in many of our major crops will continue the selection of such biotypes, and complicate weed management. There is no simple solution to managing multiple resistance in weed populations, especially highly-outcrossing species. True integrated weed management systems utilizing a number of cultural or mechanical methods and less reliance on herbicides are needed to maintain the effectiveness

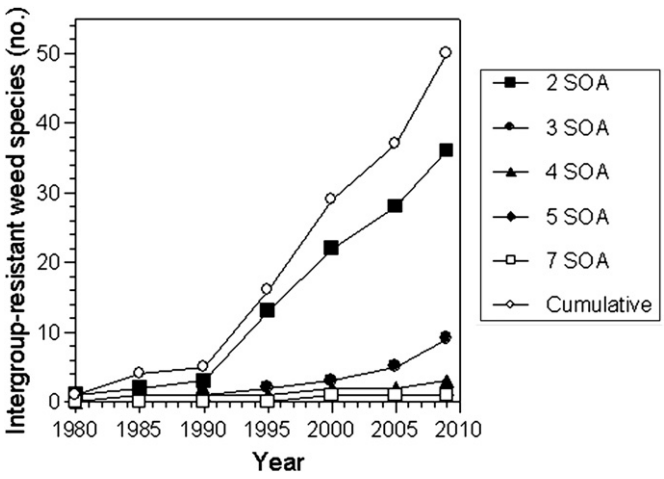

Fig. 1. Chronological increase in the number of weed species globally with intergroup herbicide resistance (SOA, site of action; compiled from Heap (2011)).

of remaining herbicide tools. Greater implementation of tactics and practices to proactively manage herbicide resistance, reviewed in Beckie (2006), is needed to mitigate the impact of intergroup-resistant weed populations.

Herbicide sequences, mixtures, and rotations generally have the greatest effect in delaying resistance when the mechanism conferring resistance is an altered target site or translocation, the target weed species are highly self-pollinated, and seed spread within and among fields is restricted (Beckie, 2006). Management of HR weed populations is markedly easier where resistance is restricted to one or more herbicides with the same site of action vs. herbicides with different sites of action. For example, glyphosate-resistant weeds are rarely cross-resistant to herbicides of other sites of action (reviewed separately in Beckie (2011)). In the following sections, cross-resistance patterns and prevalent mechanisms of resistance in weed populations to herbicides of various sites of action are summarized, and potential implications for management outlined.

2.1. Target-site resistance

2.1.1. Acetyl-CoA carboxylase (ACC) inhibitors (group A)

There are currently populations of 41 grass weed species resistant to ACC inhibitors (Heap, 2011), first commercialized in the mid- to late 1970s. Weed species or genera with high incidence of ACC-inhibitor resistance include *A. myosuroides*, and *Avena*, *Echinochloa*, *Lolium*, *Phalaris*, and *Setaria* spp. Herbicides with this site of action can select for target-site resistance in weeds in fewer than 10 applications (Beckie, 2006). Eleven target-site mutations (amino acid substitutions) in the *ACC* gene have been documented to date in populations of seven grass weed species (Table 1). Amino acid number is standardized to *A. myosuroides* plastidic homomeric *ACC*.

The level and spectrum of target-site ACC-inhibitor resistance are determined by the particular resistance mutation, homozygosity/heterozygosity of the plants for the mutation, and the herbicide and dose used for evaluation (Délye, 2005; Powles and Yu, 2010). A fitness cost has been associated with the 2078 and 2088 mutations, but not the 1781 mutation (Yu et al., 2007a; Menchari et al., 2008; Powles and Yu, 2010); lack of fitness cost for the latter mutation may explain why it apparently occurs most frequently. The Ile1781Leu mutation can confer high-level resistance to some or all herbicides in all three classes: aryloxyphenoxypropionate (APP), cyclohexanedione (CHD), and phenylpyrazolin (PPZ, pinoxaden) (Table 1). For example, biotypes with this mutation can exhibit resistance to the APP herbicides fenoxaprop, clodinafop, fluazifop, haloxyfop, etc;