



Evolution of Resistance to Auxinic Herbicides: Historical Perspectives, Mechanisms of Resistance, and Implications for Broadleaf Weed Management in Agronomic Crops

Authors: Mithila, J., Hall, J. Christopher, Johnson, William G., Kelley, Kevin B., and Riechers, Dean E.

Source: *Weed Science*, 59(4) : 445-457

Published By: Weed Science Society of America

URL: <https://doi.org/10.1614/WS-D-11-00062.1>

Evolution of Resistance to Auxinic Herbicides: Historical Perspectives, Mechanisms of Resistance, and Implications for Broadleaf Weed Management in Agronomic Crops

J. Mithila, J. Christopher Hall, William G. Johnson, Kevin B. Kelley, and Dean E. Riechers*

Auxinic herbicides are widely used for control of broadleaf weeds in cereal crops and turfgrass. These herbicides are structurally similar to the natural plant hormone auxin, and induce several of the same physiological and biochemical responses at low concentrations. After several decades of research to understand the auxin signal transduction pathway, the receptors for auxin binding and resultant biochemical and physiological responses have recently been discovered in plants. However, the precise mode of action for the auxinic herbicides is not completely understood despite their extensive use in agriculture for over six decades. Auxinic herbicide-resistant weed biotypes offer excellent model species for uncovering the mode of action as well as resistance to these compounds. Compared with other herbicide families, the incidence of resistance to auxinic herbicides is relatively low, with only 29 auxinic herbicide-resistant weed species discovered to date. The relatively low incidence of resistance to auxinic herbicides has been attributed to the presence of rare alleles imparting resistance in natural weed populations, the potential for fitness penalties due to mutations conferring resistance in weeds, and the complex mode of action of auxinic herbicides in sensitive dicot plants. This review discusses recent advances in the auxin signal transduction pathway and its relation to auxinic herbicide mode of action. Furthermore, comprehensive information about the genetics and inheritance of auxinic herbicide resistance and case studies examining mechanisms of resistance in auxinic herbicide-resistant broadleaf weed biotypes are provided. Within the context of recent findings pertaining to auxin biology and mechanisms of resistance to auxinic herbicides, agronomic implications of the evolution of resistance to these herbicides are discussed in light of new auxinic herbicide-resistant crops that will be commercialized in the near future.

Nomenclature: Auxinic herbicides; dominant trait; evolution of resistance; fitness cost; herbicide-resistant crops; mode of action; mechanism of resistance; plant growth regulator; recessive trait.

Auxinic herbicides such as 2,4-D, MCPA, and dicamba are widely used in agriculture to selectively control broadleaf weeds in cereal crops, while picloram is traditionally used for control of broadleaf weeds in nonagricultural areas (e.g., power line corridors or rangeland areas). 2,4-D and MCPA were the first group of selective organic herbicides developed during World War II, and their discovery led to a significant increase in 2,4-D production in North America (Kirby 1980). Consequently, the use of 2,4-D in cereal crops revolutionized agricultural production throughout the world. The commercial success of 2,4-D subsequently resulted in the synthesis of other compounds such as dicamba, clopyralid, picloram, and quinclorac that are currently being used as selective herbicides (Sterling and Hall 1997). Auxinic herbicides are inexpensive and do not have prolonged soil residual activity (with the exception of the pyridine carboxylic acids; Figure 1). These herbicides have been a preferred choice for weed control and the most extensively used herbicides worldwide for more than 60 yr, primarily because of their selectivity, efficacy, wide spectrum of weed control, and low application costs. The use of 2,4-D, dicamba, and other auxinic herbicides has increased in the United States, Canada, and other countries since their commercialization for row crops, as well as in noncrop systems (Industry Task Force II 2005).

Auxinic herbicides are so named because at low concentrations they mimic several physiological and biochemical effects of the natural plant hormone indole-3-acetic acid (Sterling and

Hall 1997), which is generally referred to as auxin or IAA (Went 1926). IAA plays a pivotal role in long-range signaling for systemic communication among various plant organs. Indole compounds such as indoleacetaldehyde, indole ethanol, and indoleacetonitrile, derived from the amino acid tryptophan, are the major precursors of IAA in plants (Salisbury and Ross 1992). In addition, several compounds other than IAA are also considered endogenous or natural auxins (reviewed by Simon and Petrášek 2011).

Auxinic herbicides are structurally similar to IAA (Figure 1). Based on the position of the carboxylic acid moiety and the type of aromatic group auxinic herbicides possess, these herbicides have been classified into three different classes (Ashton and Crafts 1981), viz. (1) phenoxyalkanoic acids (e.g., 2,4-D, MCPA); (2) benzoic acids (e.g., dicamba, cloramben); and (3) pyridine carboxylic acids (e.g., picloram, clopyralid, triclopyr and fluroxypyr) (Figure 1). Recently, another group of herbicides (quinolinecarboxylic acids; e.g., quinclorac and quinmerac) with auxin-like activity has been developed (Figure 1), and its members are also classified as auxinic herbicides (Grossman 2000, 2010). Alternatively, according to another scheme of classification proposed by Devine et al. (1993), one group of auxinic herbicides has an ether linkage between the aromatic group and carboxylic acid moiety (e.g., all phenoxyalkanoic acids and some pyridines; i.e., fluroxypyr and triclopyr) and in the other group the carboxylic acid is directly attached to the aromatic ring (e.g., benzoic acids and some pyridines; i.e., picloram and clopyralid). The structure-activity relationships of these groups of auxinic herbicides have been described in detail in previous reviews (Coupland 1994; Sterling and Hall 1997).

Although natural auxin—a plant hormone that influences various processes of plant growth and development (Salisbury

DOI: 10.1614/WS-D-11-00062.1

*First and second authors: Postdoctoral Research Associate and Professor, School of Environmental Sciences, University of Guelph, Ontario, Canada N1G 2W1; third author: Professor, Department of Botany and Plant Pathology, Purdue University, West Lafayette, IN 47907; fourth author: Research Scientist, AgraServ, Inc., American Falls, ID 83211; fifth author: Associate Professor, Department of Crop Sciences, University of Illinois, Urbana, IL 61801. Corresponding author's E-mail: riechers@illinois.edu

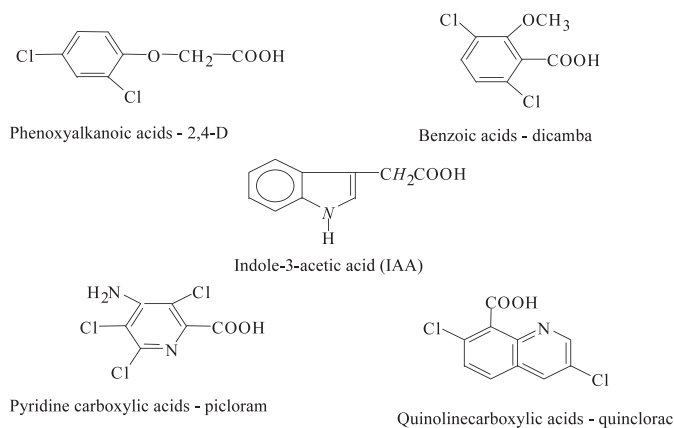


Figure 1. Chemical structures and classification of auxinic herbicides and natural auxin (IAA).

and Ross 1992)—was discovered over 90 yr ago (Went 1926), the precise mechanism of action of this hormone was uncovered only recently (Dharmasiri et al. 2005; Kepinski and Leyser 2005; Tan et al. 2007). Although the precise mode of action for auxinic herbicides remains unclear six decades after their discovery, previously published reviews describe in detail the possible receptors involved in auxinic herbicide perception, biochemical changes induced upon the auxinic herbicide application in plants, and transport of auxinic herbicides, as well as their selectivity between dicots and monocots (Coupland 1994; Kelley and Riechers 2007; Sterling and Hall 1997). Furthermore, Coupland (1994) provided detailed information about the mechanism of resistance of auxinic herbicide-resistant weed species, specifically regarding weeds that were found to be resistant to the phenoxyalkanoic acids (e.g., 2,4-D and MCPA) prior to 1994. Since 1994, several new weed biotypes resistant to a number of auxinic herbicides (i.e., clopyralid, picloram, dicamba, quinclorac, and 2,4-D) have been discovered and extensive research has been conducted to understand the mechanism of resistance in these weed biotypes. The goal of this article is to present and summarize recent advances in the auxin signal transduction pathway and its relationship to auxinic herbicide mode of action, as well as comprehensively review the literature regarding mechanisms of resistance to auxinic herbicides since the publication of the two previous extensive reviews (Coupland 1994; Sterling and Hall 1997). Importantly, the agronomic impact of resistance to these herbicides will also be discussed in light of new auxinic-resistant crop varieties that will be commercialized in the near future.

Recent Advances in Auxin Biology and Auxinic Herbicide Mode of Action

Our understanding of auxin biology, in particular auxin perception, receptor proteins, signaling, and the regulation of auxin-responsive gene expression, has advanced tremendously during the past 5 yr. Several key points from these research areas will be summarized below, and will be discussed in the context of what is currently known regarding auxinic herbicide mode of action in sensitive dicot plants.

Auxin Perception, Signaling, Gene Expression, and Homeostasis: A Tale of “Release from Repression.” New discoveries regarding auxin receptors (Dharmasiri et al. 2005; Kepinski and Leyser 2005) and subsequent modeling of the interactions between natural auxin and its receptor proteins (Tan et al. 2007) have led the way for a comprehensive molecular understanding of the role of auxin in plant growth and development. These discoveries have been recently reviewed (Calderon-Villalobos et al. 2010; Chapman and Estelle 2009; Guilfoyle 2007; Mockaitis and Estelle 2008) and thus will not be described in detail, but instead will be briefly summarized to provide a background for further discussions of auxinic herbicide mode of action and evaluating potential resistance mechanisms in weeds.

Auxins such as IAA control many plant growth and developmental processes, and accomplish this mainly due to their effects on regulating plant gene expression (Guilfoyle 2007). When auxin concentrations are low in plant tissues, auxin-responsive genes are not expressed due to the presence of Aux/IAA repressor proteins (Aux/IAAs) that bind to the promoters of auxin-responsive genes (Chapman and Estelle 2009). Following *de novo* IAA synthesis or release of IAA from stored conjugates (Ludwig-Müller 2011), auxin concentrations increase and promote gene expression by ubiquitin-mediated degradation of transcriptional repressors (Aux/IAAs), thereby activating gene expression by a novel “release from repression” mechanism. Auxins (and the synthetic auxinic herbicides) accomplish this by binding directly to the bottom of the TIR1 pocket and acting as a “molecular glue” that stabilizes the interaction between the auxin receptor protein (TIR1 and its homologs [AFBs]) and its substrates (Aux/IAA repressors) in an auxin-dependent manner (Guilfoyle 2007; Tan et al. 2007), thereby leading to rapid Aux/IAA degradation. TIR1 and related receptor proteins serve as the specificity determinant of the SCF^{TIR1} complex, which target substrate proteins (Aux/IAA repressors) for polyubiquitylation and subsequent degradation (Chapman and Estelle 2009; Mockaitis and Estelle 2008). This interaction at TIR1 leading to Aux/IAA degradation by ubiquitin and subsequent gene expression via “release from repression” is depicted in Figure 2. A “promiscuous” binding pocket on the TIR1 receptor protein tolerates binding of both natural (IAA) and synthetic (NAA or 2,4-D) auxin molecules as the “molecular glue” (Figure 2) to stabilize and strengthen the interaction between receptor protein and substrate, although IAA and 2,4-D binding occur with different affinities for TIR1 (Tan et al. 2007).

Following the rapid increase in auxin-responsive gene expression, the auxin stimulus is attenuated and transcriptional repression (via Aux/IAA binding) once again predominates within the cell (Figure 2). This is accomplished by at least two mechanisms: (1) GH3-mediated conjugation of IAA with amino acids (reviewed by Kelley and Riechers 2007; Staswick et al. 2005), or (2) Aux/IAA repressors are induced as part of the suite of primary auxin-responsive genes. As pointed out previously (Kelley and Riechers 2007), the induction of Aux/IAAs by auxins seems contradictory because they are essentially repressing their own expression. However, it is likely a mechanism that helps ensure a transient response to increased auxin levels, in which auxin-induced gene expression is quickly attenuated once the auxin stimulus is removed via auxin degradation, inactivation via conjugation reactions (Bajguz and Piotrowska 2009; Ludwig-Müller 2011; Staswick

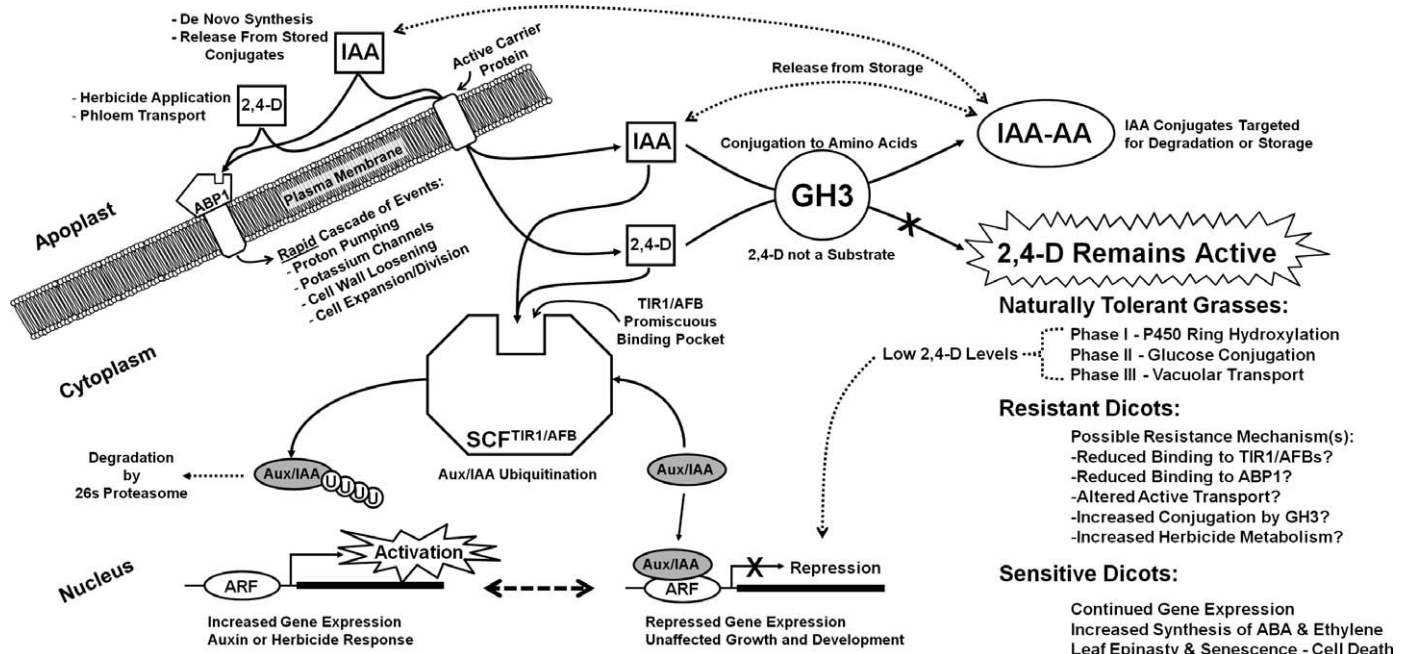


Figure 2. Proposed model depicting the sequence of events (transport, perception, and signaling) following treatment with IAA or auxinic herbicides, leading to both gene induction (via release from repression; i.e., degradation of Aux/IAs) and subsequent return to “normal” conditions (homeostasis) in sensitive dicots, tolerant grasses, and resistant dicot weeds. Note that tolerant grasses and resistant dicot weeds may have different mechanisms for dealing with the unregulated auxin response, or in some cases may avoid it entirely. Please refer to Grossman (2010) for a more detailed explanation of the cascade of processes leading to plant death in sensitive dicots. Abbreviations and terms used in the Figure: ABP1, auxin-binding protein 1; Aux/IAs, auxin/indole-3-acetic acid transcriptional repressors of auxin-regulated gene expression, but are induced by high auxin concentrations as a feedback mechanism; ARFs, pre-existing, DNA-binding auxin-response proteins that activate gene transcription in response to high auxin concentrations; AFBs, auxin signaling F-box protein (homologous to TIR1, see below); GH3 protein, enzyme that reversibly conjugates IAA with amino acids to form an inactive conjugate; SCF, a complex consisting of Skp1-cullin-F-box proteins; SCF^{TIR1}, SCF-ubiquitin ligase E3 complex containing the TIR1 protein, which is part of the ubiquitin-26S proteasome-mediated pathway for protein degradation and turnover; TIR1, transport inhibitor response1 protein (homologous to AFBs), which is the specificity determinant of the SCF^{TIR1} complex that targets substrate proteins (Aux/IAA repressors) for polyubiquitylation and subsequent degradation.

2009), reduced biosynthesis, or sequestration (Kelley and Riechers 2007). Interested readers are referred to a recent comprehensive study (Petersson et al. 2009) for a more detailed discussion of the factors contributing to auxin homeostasis in plant root cells and tissues.

The entire pathway ranging from application of an auxinic herbicide (such as 2,4-D) to a plant, to death in sensitive dicots, tolerance in grasses, or resistance in dicots, is depicted in Figure 2. Of particular significance to the mechanism of action of auxinic herbicides in sensitive dicots are four steps in the entire sequence of events: (1) both IAA and 2,4-D are able to bind to the “promiscuous pocket” of TIR1 or its homologs, leading to auxin-responsive gene expression, (2) both IAA and 2,4-D are actively transported into plant cells via a common carrier protein (see discussion below), (3) IAA is a substrate for conjugation by the GH3 family of proteins but 2,4-D is not a GH3 substrate, and (4) 2,4-D is not ring hydroxylated by cytochrome P450s or other metabolic enzymes in sensitive dicots. The fact that auxinic herbicides are substrates for proteins/enzymes in these first two steps (i.e., signaling and transport) and not substrates for enzymes in the final two steps (i.e., conjugation and detoxification) are likely responsible for their lethality in sensitive dicots (Kelley et al. 2004; Kelley and Riechers 2007); however, they might also contribute as potential resistance mechanisms in auxinic herbicide-resistant dicot weeds if mutations in these proteins reduce herbicide binding (steps 1 and 2) or increase binding of auxinic herbicides (steps 3 and 4) (Figure 2). These

potential mechanisms of resistance in dicot weeds and fitness penalties that may result from these mutations will be further discussed in detail later in this review.

It should also be noted that the plasma membrane-localized auxin-binding protein 1 (ABP1) is involved in a broad range of cellular responses to auxin (such as cell expansion, cell division, and regulating ion fluxes at the plasmalemma), as well as auxin-regulated gene expression (Figure 2) (reviewed by Tromas et al. 2010). ABP1 is different from TIR1 and its homologs because it is a membrane-bound receptor for auxin-mediated signaling (Tromas et al. 2010), which is in contrast to the soluble (cytosolic) auxin receptor TIR1. The different roles and functions of ABP1 in mediated responses to natural auxins or its possible relevance in auxinic herbicide mechanism of action in plants will not be discussed here in detail, but it is important to note that ABP1 has been implicated as a potential target site that confers resistance in several auxinic herbicide-resistant dicot weeds (further discussed below).

Auxinic Herbicide Physiology, Cellular Transport, and Mode of Action. One of the few published reports that directly link auxinic herbicide mode of action with the recent discovery of auxin receptors was reported by Walsh et al. (2006). This study reported the generation and analysis of mutants impaired in auxin signaling in *Arabidopsis*, and discussed how these findings may affect cross resistance to different auxinic herbicide subclasses (i.e., phenoxyacetic acids vs. pyridine carboxylic acids; Figure 1). Importantly, it was

Review of Auxinic Herbicide Resistance in Weeds

discovered in their mutant screen that several alleles at two distinct genetic loci confer resistance to picloram but not to 2,4-D or IAA. One picloram-specific resistance locus encoded auxin-signaling F-box protein 5 (AFB5), which is one of the five homologs of TIR1 in the *Arabidopsis* genome (Tan et al. 2007; Walsh et al. 2006). An additional mutant of interest (*axr1-3*) was identified that conferred very high levels of cross resistance to picloram, 2,4-D, and an experimental auxinic herbicide compound (Walsh et al. 2006), indicating that the different auxin receptors in plants may be specific or general and mutations may confer different patterns of cross resistance to the various subclasses of auxinic herbicides (Figure 1). These authors speculated that the functional redundancy in auxin receptors (i.e., TIR1 and AFBs 1–5) may contribute to the relatively low abundance of auxinic herbicide-resistant weeds (i.e., multiple sites of action for auxinic herbicides in plant genomes), as well as potential for fitness penalties that may occur due to mutations in the auxin signaling pathway (this aspect is further discussed below). It is also important to note that the role of ABP1 as an auxin receptor is not as clearly defined as the TIR1/AFBs regarding natural or synthetic auxins (as noted above), but that ABPs may also play a role in weed resistance to auxinic herbicides.

In addition to auxin receptors that initiate auxin-mediated signaling, another similarity between natural and synthetic auxins is that 2,4-D is taken up by a carrier-mediated, active transport mechanism in plant cells (Figure 2) (Sterling 1994), which is presumably the same carrier that transports IAA (Rubery 1977). This transport protein could present a novel mechanism for auxinic herbicide resistance (i.e., altered cellular transport or exclusion) in weeds, similar to glyphosate resistance in horseweed [*Conyza canadensis* (L.) Cronq] via increased vacuolar transport and sequestration (Ge et al. 2010). Active, carrier-mediated transport of herbicides into cells is not common; most herbicides enter cells via passive diffusion, or for weak acids by an ion trapping mechanism (Sterling 1994). Glyphosate (via a phosphate carrier), paraquat (via polyamine carriers), and 2,4-D (via IAA influx/efflux carriers) are notable exceptions among herbicides (Sterling 1994). As noted above, it could be possible that a mutation preventing 2,4-D or other auxinic herbicides from being recognized by the IAA carrier protein, yet maintaining IAA as a substrate, could lead to weed resistance by excluding the herbicide from entering the cell or preventing its long-distance transport within the plant (Figure 2).

Recent findings by Grossman and colleagues (Grossman 2000, 2010; Grossman and Hansen 2001; Grossman et al. 1996) indicate the sensitive dicots do not only “grow themselves to death” due to the unregulated auxin response (Figure 2), but that lethality and necrosis in sensitive plants is actually due to the hyperaccumulation of ethylene, ABA, and reactive oxygen species (ROS) in addition to unregulated auxin activity in herbicide-treated plants (Romero-Puertas et al. 2004). The detailed time course of events in relation to hyperaccumulation of these hormones or defense compounds, the enzymes involved in their syntheses, and their combined effects on lethality, senescence, and necrosis in sensitive dicots have been thoroughly discussed in a recent review (Grossman 2010). It should also be noted that accumulation of cyanide in response to the auxinic herbicide quinclorac in sensitive grasses as part of its mode of action has been described in detail by Grossman (2010).

History and Evolution of Resistance. Generally, the occurrence of resistant plants increases slowly in the presence of continued herbicide application (Devine and Shukla 2000). Thus, herbicide selection pressure is considered as one of the primary causes for the evolution of herbicide-resistant biotypes. Prolonged use of herbicides (and resulting selection pressure) has resulted in the development of resistance to several classes of herbicides, including auxinics. However, the incidence of auxinic herbicide resistance is relatively low, as it has been reported in only 29 weed species (Heap 2011; Table 1). Since the discovery of the first auxinic herbicide-resistant wild carrot (*Daucus carota* L.) biotype in Canada, there has been a steady but slow increase in the number of auxinic herbicide-resistant weeds worldwide (Table 1). Based on the data presented in Table 1, it is important to note the large increase in the number of auxinic herbicide-resistant weeds discovered after the 1980s, with the highest incidence of cases (11) recorded between 1990 and 1999. On the other hand, relatively few auxinic herbicide-resistant weeds (six) were reported during the prior decade (from 2000 to 2010; Heap 2011).

Despite the frequent and repeated usage of auxinic herbicides in the lawncare and turfgrass industries for POST broadleaf weed control, there are few reports of auxinic herbicide-resistant weed biotypes that have been selected for under these conditions that appear to be ideal for evolution of resistance. The peculiar lack of weed resistance in these situations may be attributed to several factors, such as (1) usually two or more auxinic herbicides are tank mixed, and rarely is an auxinic herbicide from only one subfamily applied, (2) use of high seeding rates and the stoloniferous/rhizomatous growth habit of turf results in a dense sward of grass, which suppresses weed seed emergence and also reduces weed competition, (3) regular fertilization regimes and mowing contribute to reduced weed competition, (4) higher spray volumes are used to obtain optimal coverage in turfgrass and high doses of herbicides are frequently used, and (5) hand weeding or other nonchemical methods are used to eliminate hard-to-control weeds. These factors, combined with prudent management practices, may have resulted in reduced incidences of weed resistance to auxinic herbicides in turf. However, unlike in field crops, there is little or no monitoring for weed resistance in turfgrass. The occurrence of resistance in turfgrass settings may thus be greatly underestimated, or it may simply be due to a lack of concerted efforts to investigate cases of poor weed control since the economic cost to homeowners is minimal compared with the impact of crop losses on a farmer's livelihood.

Genetics and Inheritance of Weed Resistance. The rarity in occurrence of auxinic herbicide resistance compared with other herbicide classes (such as acetolactate synthase [ALS] inhibitors or *s*-triazines; Heap 2011) has been attributed to proposed multiple sites of action of these compounds (Gressel and Segel 1982; Morrison and Devine 1994). However, this hypothesis has not been tested directly. To understand the evolution of resistance to auxinic herbicides it is important to investigate the nature of inheritance of the resistance trait. Compared with crop plants, where inheritance of economically important traits has been investigated in a large number of species, there are relatively few studies describing the

Table 1. Chronological listing of auxinic herbicide-resistant weeds^a discovered to date (listed by decade).

1950–1979		1980–1989		1990–1999		2000–2010	
Species	Country (Year)	Species	Country (Year)	Species	Country (Year)	Species	Country (Year)
Wild carrot	Canada (1957 ^b)	<i>Carduus nutans</i> (Musk thistle)	New Zealand (1981)	Wild mustard	Canada (1990)	<i>Echinochloa colona</i> (Junglerice)	Colombia (2000)
Spreading dayflower	USA (1957)	<i>Sphenoclea zeylanica</i> (Gooseweed)	Philippines (1983)	<i>Papaver rhoeas</i> (Corn poppy)	Spain (1993)	<i>Limnophila erecta</i> (Marshweed)	Malaysia (2002)
Field bindweed	USA (1964)	<i>Stellaria media</i> (Common chickweed)	United Kingdom (1985)	Kochia	USA (1995)	<i>Digitaria ischaemum</i> (Smooth crabgrass)	USA (2002)
<i>Matricaria perforate</i> (Scentless chamomile)	France (1975)	Tall buttercup	New Zealand (1988)	<i>Limnocharis flava</i> (Yellow bur-head)	Indonesia (1995)	<i>Cenopodium album</i> (Lambsquarters)	New Zealand (2005)
<i>Cirsium arvense</i> (Canada thistle)	Sweden (1979)	Yellow starthistle	USA (1988)	False cleavers	Canada (1996)	<i>Sisymbrium orientale</i> (Indian hedge mustard)	Australia (2005)
		<i>Fimbristylis miliacea</i> (Globe fringerush)	Malaysia (1989)	<i>Carduus pycnocephalus</i> (Italian thistle)	New Zealand (1997)	Prickly lettuce	USA (2007)
				Common hempnettle	Canada (1998)	<i>Salsola iberica</i> ^c (Russian thistle)	USA (2011)
				<i>Echinochloa crus-galli</i> (Barnyardgrass)	USA (1998)		
				<i>Echinochloa crus-pavonis</i> (Gulf cockspur)	Brazil (1999)		
				<i>Raphanus raphanistrum</i> (Wild radish)	Australia (1999)		
				<i>Soliva sessilis</i> (Carpet burweed)	New Zealand (1999)		

^a Data are from Heap (2011) <weeds-science.org>.

^b Reported by Dr. Clay Switzer, University of Guelph (personal communication).

^c Reported by Dr. William Dyer, Montana State University (personal communication).

inheritance of herbicide resistance in weed species. This is not due to our lack of understanding of plant genetics, but rather due to the paucity of information available to assist in the genetic analysis of herbicide resistance (Darmency 1994). Nonetheless, it has been demonstrated that inheritance of herbicide resistance to the majority of herbicide families (including auxinic herbicides) is controlled by a single gene, or in fewer cases, by two major genes (Preston and Mallory-Smith 2001).

Studies on inheritance of auxinic herbicide resistance in weed species demonstrate that dicamba, 2,4-D, and picloram resistance in wild mustard (*Sinapis arvensis* L.) is conferred by a single, dominant gene (Jasieniuk et al. 1995; Jugulam et al. 2005). In addition, dicamba resistance in kochia (*Kochia scoparia* L.; biotypes from Henry, Nebraska) is determined by a single allele with a high degree of dominance (Preston et al. 2009). Conversely, a single recessive gene controls clopyralid and picloram resistance in yellow starthistle (*Centaurea solstitialis* L.) (Sabba et al. 2003) and quinclorac resistance in false cleavers (*Galium spurium* L.) (Van Eerd et al. 2004). It has been reported that two additive genes control MCPA resistance in common hempnettle (*Galeopsis tetrahit* L.) (Weinberg et al. 2006).

If a resistance trait is determined by a single or few major genes, the rate at which the trait can spread within a population is much higher compared with a polygenic trait. Since the chance of simultaneous occurrences of several mutations leading to resistance in a single plant is a product of its individual probabilities, the possibility of a polygenic trait occurring is much lower than for a qualitative trait (Jasieniuk et al. 1996). Furthermore, inheritance of polygenic traits depends on outcrossing for accumulation of several genes with minor effects, which in turn reduces the evolution of herbicide resistance. Accordingly, Gressel and Segel (1982) suggested

that the low incidence of auxinic herbicide resistance may be due to the requirement of mutations at several loci in order to impart resistance, hence the postulation of multiple modes of action of auxinic herbicides in dicot weeds (Gressel and Segel 1982). It appears that this may not be the case because, as described above, many auxinic herbicide-resistance traits are conferred by a single gene. Despite the long history of auxinic herbicide use, the relatively low incidence of resistant biotypes and their inability to evolve and propagate in agronomic crops may be due to the rare occurrence of resistant individuals (and resistance alleles) in natural weed populations, or that mutations conferring resistance are lethal (Jasieniuk et al. 1995). It is also possible that the relatively low selection pressure or short-lived residual activity (except for the pyridines; Figure 1) in the soil may contribute to the low occurrence of auxinic herbicide-resistant weeds.

Fitness Costs Associated with Weed Resistance. According to the theory of natural selection, herbicide-resistant plants are already present in the population before the use of the herbicide; thus, herbicides do not directly cause genetic mutations imparting resistance. Natural mutations resulting in herbicide resistance in populations may be associated with a fitness cost, either due to pleiotropic effects of the resistance gene itself or due to linkage of the resistance gene with one or more other loci that impose the fitness cost. Information about fitness costs associated with auxinic herbicide resistance has been documented in only a few weed species. Bourdot et al. (1996) reported that when grown together, MCPA-resistant giant buttercup (*Ranunculus acris* L.) plants are less competitive and ecologically less fit than sensitive plants in the absence of herbicide application. However, competitiveness varied under different plant densities. Both resistant and sensitive plants yielded similarly at low densities, whereas

sensitive plants yielded more at higher plant densities. Similarly, analyses of morphological traits (plant height, leaf area, and root system; Hall and Romano 1995) and seed yield (Debreuil et al. 1996) between auxinic herbicide-resistant and -sensitive wild mustard plants indicate that resistant plants are less fit in the absence of herbicide application. Fitness penalties associated with auxinic herbicide resistance (Bourdot et al. 1996; Debreuil et al. 1996; Hall and Romano 1995) may be a major reason for the limited occurrence of auxinic-herbicide resistant weeds (Walsh et al. 2006).

When herbicide resistant and sensitive plants arise from different populations, they may exhibit genetic variability at various loci related to plant fitness (Jasieniuk et al. 1996). In order to attribute fitness costs unequivocally, it is important to conduct fitness studies using herbicide resistant and sensitive plants with similar genetic backgrounds differing only for the alleles imparting herbicide resistance (Vila-Aiub et al. 2009), such as in near-isogenic lines (NILs) (Jasieniuk et al. 1996). Thus far, fitness studies using NILs of weed species have only been performed with triazine-resistant and -sensitive lines (Beverdorf et al. 1988; Gressel and Ben-Sinai 1989; Jacob et al. 1988; McCloskey and Holt 1991). These studies clearly showed that the most common mutation in the D1 protein resulting in triazine resistance (serine 264 to glycine) is associated with impaired photosynthetic electron transport and reduced plant fitness. Experiments are in progress (J. C. Hall, unpublished data) to assess fitness costs associated with auxinic herbicide resistance in wild mustard using NILs.

Mechanisms of Weed Resistance to Auxinic Herbicides.

Although 29 auxinic herbicide-resistant weeds have been discovered to date (Heap 2011; Table 1), the characterization of mechanisms of resistance has been investigated in only some of these weeds. Coupland (1994) reviewed the differences between auxinic herbicide-resistant and -sensitive biotypes of several weed species in terms of their morphological differences, as well as their levels of resistance. However, since 1994 a number of auxinic herbicide-resistant weeds with unknown mechanisms of resistance have been investigated to identify differences in herbicide uptake, translocation, metabolism, or potential target site alterations. Recently, 2,4-D-resistant prickly lettuce (*Lactuca serriola* L.) biotypes were determined to be 25-fold resistant compared with sensitive biotypes in a dose-response experiment (Burke et al. 2009; Singh 2009). However, the mechanism of 2,4-D resistance in this biotype has not been reported.

Wild Mustard. Among the auxinic herbicide-resistant weeds identified to date, the mechanism of auxinic herbicide resistance in wild mustard has been most extensively characterized and comprehensively reviewed (Hall et al. 1996; Zheng and Hall 2001). Based on dose-response experiments conducted in growth chambers, the resistant biotypes of wild mustard are 10-, 18-, and 104-fold resistant to MCPA, 2,4-D, and dicamba, respectively, relative to sensitive biotypes (Heap and Morrison 1992). Resistance to auxinic herbicides in wild mustard is not due to altered uptake, translocation, or metabolism (Peniuk et al. 1993). However, when treated with picloram, the sensitive biotypes produced more ethylene than resistant biotypes (Hall et al. 1993; Peniuk et al. 1993). This was attributed to differences at the primary site of action resulting in reduced expression of ACC synthase (a key enzyme

in the ethylene biosynthetic pathway; Grossman 2000, 2010), concomitant with auxinic herbicide treatment in the resistant biotype (Hall et al. 1993; Wei et al. 2000). Subsequently, a number of experiments were conducted to investigate the role of ABP1 in auxinic herbicide resistance in wild mustard. ABP1 in the sensitive biotype possessed both low and high affinity-binding sites, whereas ABP1 in the resistant biotype had only a low affinity-binding site (Webb and Hall 1995). Furthermore, upon treatment with picloram, ABP1 antisense (tobacco) and overexpressing (*Arabidopsis*) lines displayed several physiological responses that are analogous to auxinic herbicide-resistant and sensitive wild mustard biotypes, respectively (Mithila and Hall 2005). However, these studies do not conclusively provide evidence that auxinic herbicide resistance in wild mustard is due to an altered ABP1 homolog.

Recently, in an effort to conclusively identify the auxinic herbicide resistance gene in wild mustard, molecular and morphological markers linked to this trait were identified. A morphological marker for leaf shape linked to the auxinic herbicide resistance gene was identified, and several molecular markers linked to the auxinic herbicide resistance locus were identified by amplified fragment length polymorphism analysis (Mithila et al., unpublished data). Consequently, a genetic map of this locus was constructed that may assist in future cloning and identification of the auxinic resistance gene in wild mustard (Mithila et al., unpublished data).

Kochia. The physiological, biochemical, and molecular basis for dicamba resistance in kochia (biotypes from Montana) has been studied extensively (Cranston et al. 2001; Dyer et al. 2002; Kern et al. 2005). It was initially reported that resistance to dicamba was not due to altered rates of herbicide uptake, translocation, or metabolism in kochia (Cranston et al. 2001). It was also suggested that a mutation in the auxin receptor(s) may affect endogenous auxin binding and alter auxin-mediated responses, such as gravitropism and root growth inhibition (Dyer et al. 2002; Goss and Dyer 2003). Furthermore, differential gene expression patterns indicate that several transcripts were up- or down-regulated by dicamba treatment (Kern et al. 2005). In addition to induction of ACC synthase transcripts (involved in ethylene synthesis; Grossman 2000, 2010), up-regulation of a putative chloride channel protein and an unknown gene were detected. Whether or not these genes/proteins are directly involved in conferring the resistant phenotype in kochia or are expressed as a secondary consequence of the mode of action of dicamba remains to be determined, but these results provide a useful starting point for further metabolomic or proteomic (Zhang and Riechers 2008) studies in kochia.

False Cleavers. Investigation of the physiological and biochemical basis of quinclorac-resistant in false cleavers suggests that the mechanism of quinclorac resistance is not due to a difference in absorption, translocation, root exudation, or metabolism (Van Eerd et al. 2005). A significant increase in both ethylene and ABA levels was observed upon quinclorac application in sensitive biotypes but not in resistant biotypes. These authors suggested that a potential alteration in auxin signal transduction pathway might impart resistance to quinclorac in resistant biotypes. Quinclorac also controls some grass weeds and cases of resistance to this herbicide in barnyardgrass and crabgrass have been observed (Abdullah et al. 2006; Lovelace et al. 2007). A recent review by

Grossman (2010) discusses the role of cyanide accumulation in response to quinclorac treatment in great detail.

Yellow Starthistle. Clopyralid- and picloram-resistant yellow starthistle biotypes were discovered in the United States in 1988 (Table 1). The mechanism of selective resistance to pyridine herbicides is not due to differential uptake, translocation, or metabolism (Fuerst et al. 1996; Valenzuela et al. 2001) and currently remains unknown. Upon application of clopyralid or picloram, sensitive biotypes produced more ethylene than resistant types, although this response was not attributed as the major causal factor in conferring resistance in yellow starthistle (Sabba et al. 1998; Valenzuela et al. 2002) and may instead be a secondary consequence of senescence and lethality in sensitive biotypes (Grossman 2000, 2010). It has been postulated that the recessive mutation conferring pyridine-specific resistance in these yellow starthistle biotypes (Sabba et al. 2003) may be due to an altered auxin receptor (AFB), similar to that identified in picloram-resistant *Arabidopsis* mutants (Walsh et al. 2006).

Wild Radish. Wild radish populations resistant to 2,4-D and MCPA (but not to dicamba) were recently discovered in a crop rotation of lupin (*Lupinus angustifolius* L.) and wheat (*Triticum aestivum* L.) in the northern region of Western Australia (Walsh et al. 2004). Alternating herbicide treatments, including glyphosate, sulfonylureas, and 2,4-D amine (in wheat) and *s*-triazines/diflufenican (in lupin), were applied to these fields for more than 17 yr. A minimum of two selective herbicides were applied to these fields per year during this period. These wild radish populations exhibit multiple resistances to herbicides with at least four modes of action (Walsh et al. 2004), including (1) a phenoxy-, phytoene desaturase-, and triazine-resistant biotype, and (2) a phenoxy-, phytoene desaturase-, and ALS-resistant biotype.

These phenoxy-resistant wild radish populations (Walsh et al. 2004) were used in an experiment to study the growth and reproductive ability of these biotypes, with or without 2,4-D amine treatment, when growing in competition with wheat (Walsh et al. 2009). Higher densities of wheat combined with 2,4-D amine treatment resulted in satisfactory weed control, lower plant biomass, and reduced seed output in some of the phenoxy-resistant wild radish populations from Australia. However, the authors cautioned that although resistance levels in these wild radish populations are relatively low (e.g., about 2.5-fold resistant to 2,4-D amine; Walsh et al. 2004) and resistant plants are adversely affected by treatment with a recommended field-use rate of 2,4-D, continuous long-term use of phenoxy herbicides may increase the frequency of phenoxy-resistant wild radish populations in Australian wheat fields or increase the magnitude of phenoxy resistance in individual biotypes (Walsh et al. 2009).

Hempnettle. MCPA-resistant and sensitive common hempnettle biotypes evolved in a field in Alberta, Canada following repeated applications of MCPA in herbicide mixtures (Table 1). The physiological basis of MCPA resistance in common hempnettle has been investigated (Weinberg et al. 2006). MCPA resistance is not due to differences in herbicide absorption, but was attributed to a lower amount of translocation and a higher rate of metabolism in roots (Weinberg et al. 2006).

Table 2. The five most commonly cited weeds in North Central Weed Science Society presentation titles between 1948 and 2007.

Rank	1948	1955	1967	1972	1977	1982	1987	1992	1997	2002	2007
1	Canada thistle (<i>Cirsium arvense</i>)	Canada thistle	Field bindweed	Canada thistle	Common milkweed (<i>Asclepias syriaca</i>)	Eastern black nightshade (<i>Solanum pychanthum</i>)	Canada thistle	Canada thistle	Common sunflower (<i>Helianthus annuus</i>)	Common sunflower	Common lambsquarters (<i>Chenopodium album</i>)
2	Field bindweed	Field bindweed	Western ironweed (<i>Vernonia baldwinii</i>)	Field bindweed	Field bindweed	Giant foxtail	Downy brome (<i>Bromus tectorum</i>)	Common ragweed	Common waterhemp (<i>Amaranthus rudis</i>)	Common waterhemp	Common waterhemp
3	Leafy spurge (<i>Euphorbia esula</i>)	Large crabgrass	Wild buckwheat (<i>Polygonum convolvulus</i>)	Johnsongrass (<i>Sorghum halepense</i>)	Johnsongrass	Johnsongrass	Musk thistle (<i>Carduus nutans</i>)	Giant foxtail	Giant foxtail	Giant foxtail	Giant ragweed
4	Perennial sow thistle (<i>Sonchus arvensis</i>)	Leafy spurge	Wild oat	Wild oat	Wild buckwheat	Quackgrass	Velvetleaf (<i>Abutilon theophrasti</i>)	Johnsongrass	Palmer amaranth	Horseweed	Horseweed
5	Russian knapweed (<i>Acroptilon repens</i>)	Quackgrass	Yellow nutsedge (<i>Cyperus esculentus</i>)	Yellow nutsedge	Yellow nutsedge	Wild proso millet	Woolly cupgrass (<i>Eriochloa villosa</i>)	Velvetleaf	Velvetleaf	Velvetleaf	Volunteer corn (<i>Zea mays</i>)

Influence of Herbicides and Herbicide-Resistant Crops on Weed Populations

Impacts of 2,4-D, Triazines, ALS Inhibitors, and Glyphosate on Weed Populations. Reliance on a single herbicide has resulted in weed shifts since the introduction of selective herbicides in the 1940s. Harper (1956) was the first to seriously consider population changes imposed by herbicides. 2,4-D was very effective in control of broadleaf weeds, especially in monocot crops, but because of this specificity, the use of 2,4-D alone was not sufficient for protecting yields in crops that were infested with both grass and broadleaf weeds. In 1948, just 1 yr after commercialization of 2,4-D, Lee (1948) stated, "It is apparent that the use of 2,4-D alone is not the answer to weed control in corn fields. Cultivation is necessary to destroy grassy weeds." This may have been one of the earliest documentations describing a weed shift due to herbicide use, and the importance of herbicide and tillage integration. In production fields where 2,4-D was used annually, grasses dominated weed communities by the 1950s. Weed research in the Midwestern U.S. reflected these weed shifts, with much research in the 1950s and 1960s focusing on large crabgrass [*Digitaria sanguinalis* (L.) Scop.], wild oat (*Avena fatua* L.), and quackgrass [*Elytrigia repens* (L.) Desv. Ex. B.D. Jackson] control (Table 2). Repeated use of 2,4-D in Hawaiian sugarcane production raised questions about the evolution of herbicide resistance in weeds in the late 1950s. Hanson (1962) reported biotypic differences in 2,4-D sensitivity in spreading dayflower (*Commelina diffusa* Burm. f.) following use of 2,4-D in Hawaii sugarcane production. Ultimately, that population was not considered "2,4-D-resistant," but selectivity for shifting weed populations to herbicide-resistant individuals was clearly demonstrated.

The introduction of the triazine herbicides in the 1950s led to the rapid evolution of triazine-resistant weeds and a shift in weed populations to redroot pigweed (*Amaranthus retroflexus* L.) and fall panicum (*Panicum dichotomiflorum* Michx.), which germinate relatively late in the growing season (Triplett and Lytle 1972). Today there are 69 weed biotypes resistant to triazine herbicides (Heap 2011). Another major advancement in herbicide discovery was the commercialization of the ALS inhibitor herbicides in the 1980s. These herbicides were adopted rapidly in wheat, corn, and soybean. However, ALS-resistant weed biotypes and populations developed rapidly and currently there are over 100 ALS-resistant weed species worldwide (Heap 2011). Weed species that are difficult to control with glyphosate have become more common, and the evolution of glyphosate-resistant (GR) weeds, currently 10 species in the United States and 21 worldwide, continues to increase (Heap 2011; Owen and Zelaya 2005; Powles and Preston 2006).

In spite of these shifts in weed populations, 2,4-D was used on 21 to 33% of U.S. wheat, corn, cotton, and soybean hectares in 2004, atrazine was used on 64% of U.S. corn hectares in 2005, ALS inhibitors were used on 35% of U.S. soybean hectares in 2006, and glyphosate continues to be used on the vast majority of corn, soybean, and cotton hectares in the United States (United States Department of Agriculture - National Agricultural Statistics Service 2008). In summary, species shifts and development of resistant weed populations have not rendered these herbicides obsolete; however, there is increasing concern of weed shifts and resistance evolution in

GR crops. It is therefore important to review the literature pertaining to how previous weed management and recent agronomic practices have impacted weeds and their composition in natural populations. This information can be used to gain a greater understanding of how weed population dynamics may change when 2,4-D and dicamba-resistant crops are introduced within the next 5 to 10 yr.

Impact of Glyphosate-Resistant Crops. GR crops, first released in 1996, have been the most rapidly adopted agriculture technology by farmers in the United States. When GR crops were introduced, it was postulated that resistance due to an altered, insensitive target site enzyme for glyphosate was highly unlikely because of the extremely complex manipulations that were required to develop GR crops (Bradshaw et al. 1997). Other scientists suggested that use of glyphosate would inevitably cause shifts in weed species under continual selection pressure from the herbicide (Duke 1996; Shaner 2000). As noted below, several cases of weed shifts have been documented since the introduction of GR soybean, cotton, and corn in the 1990s, likely due to the intense selection pressure from relying on glyphosate and the concomitant reduction in the amount of tillage used in agronomic crops (Culpepper 2006).

Firbank et al. (2006) documented that fields of GR corn had higher weed seedbank densities in the first 2 yr of GR use compared with a conventional herbicide system. Jeschke and Stoltenberg (2006) found that 8 yr of continuous glyphosate used alone in a corn-soybean rotation resulted in greater weed species richness compared to more diverse management systems that included tillage or nonglyphosate herbicides. For example, weed species composition during these 8 yr included common lambsquarters, pigweed (*Amaranthus*) species, and giant foxtail (*Setaria faberi* Herrm.), which predominated across most treatments. However, treatments consisting of only one POST application of glyphosate were dominated by giant ragweed (*Ambrosia trifida* L.), shattercane, and large crabgrass. Field survey research in Indiana has shown that GR horseweed, giant ragweed, and tolerant common lambsquarters populations were among the most prevalent among late-season weed escapes in Indiana soybean fields in 2003, 2004, and 2005 (Davis et al. 2008).

Hilgenfeld et al. (2004) conducted a 2-yr, multi-site study on weed species shifts in GR soybean systems. This study showed that continuous use of glyphosate for weed management altered the presence of weed species found within a given field. This was due to the inconsistent control of all the species and allowing some species such as ivyleaf morningglory [*Ipomoea hederacea* (L.) Jacq.] and shattercane [*Sorghum bicolor* (L.) Moench] to disproportionately replenish the seedbank. Wilson et al. (2007) found that during a 5-yr period in glyphosate-based cropping systems in the western U.S. Corn Belt, weed populations shifted from kochia (*K. scoparia* L. Schrad.) and wild-proso millet (*Panicum miliaceum* L.)-dominated populations to a predominately narrowleaf lambsquarters (*Chenopodium desiccatum* A. Nels.) population. In summary, increased glyphosate use since 1996 has shifted populations to competitive and difficult-to-control weeds such as giant ragweed, horseweed, common and narrowleaf lambsquarters, morningglory, and shattercane (Davis et al. 2008; Hilgenfeld et al. 2004; Jeschke and Stoltenberg 2006; Wilson et al. 2007).

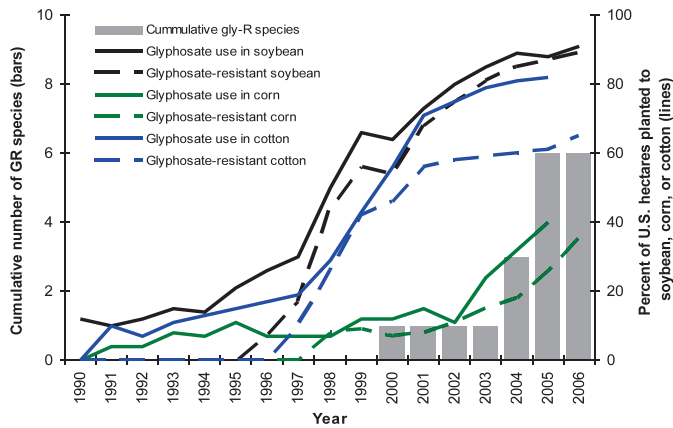


Figure 3. Rate of glyphosate-resistant (GR) crop adoption and glyphosate use in the United States compared with the number of weeds that have evolved resistance in GR cropping systems in the United States.

It has also been of great interest to track the weeds that weed scientists were conducting research on during the course of time in the Midwest. From 1948 until the early 1980s, perennial weeds were the predominant weed species under investigation in the U.S. Corn Belt (Table 2). By 1997, perennial weeds were not among the top five most commonly cited weeds in North Central Weed Science Society (NCWSS) Annual Meeting Proceedings. By 2007, the top five most commonly cited weeds in these Proceedings had either evolved resistance to glyphosate, contained an herbicide-resistance gene (i.e., volunteer GR corn), or are naturally tolerant to glyphosate (Table 2). Prior to GR crop introduction there were no reports of weeds resistant to glyphosate (Gustafson 2008); however, since the introduction of GR soybean in 1996, glyphosate resistance has been well documented (Heap 2011). The evolution of GR weeds has been closely associated with the frequency of glyphosate use for selective weed management in agronomic crops (Figure 3). In addition, results from grower surveys (Childs et al. 1997; Kruger et al. 2008; Nice and Johnson 2005) indicate broadleaf weeds that are either resistant or difficult to control with glyphosate have become more

problematic than monocot species since the introduction of GR cropping systems (Table 3).

Evolution and Impact of Glyphosate Resistance in Weeds.

Several cases of weeds that vary in their sensitivity to glyphosate have been reported since it was first introduced in the 1970s. For example, DeGennaro and Weller (1984) identified field bindweed (*Convolvulus arvensis* L.) biotypes that varied up to 70% in visual injury, root and shoot dry weight, and shoot regrowth when exposed to a 2.2 kg ha⁻¹ application of glyphosate. Furthermore, as much as a 40% increase in tolerance to glyphosate was observed as the plants matured.

Horseweed was the third GR weed identified worldwide, but was the first GR weed documented to have evolved in a GR cropping system in Delaware (VanGessel 2001). Since this initial report, GR horseweed has been reported in at least 15 other states from Delaware to California and from Michigan to Mississippi (Heap 2011). GR horseweed has been reported in other countries including Brazil, China, Spain, and the Czech Republic (Heap 2011). An Indiana survey conducted from 2003 to 2005 (Davis et al. 2008) showed the frequency of GR horseweed was as high as 38% in all Indiana soybean fields in the southeast region of the state and infested over 100,000 hectares surveyed statewide. As evidenced by this high frequency in some geographies, GR horseweed may be a compounding problem for large geographies because in-field and seedbank demographics were shown to increase in systems that rely on glyphosate (Davis et al. 2007) and seeds are easily transported long distances by wind (Dauer et al. 2006).

Other GR weeds that evolved in the United States in GR crops include common ragweed (*Ambrosia artemisiifolia* L.), giant ragweed, common waterhemp, and Palmer amaranth (*Amaranthus palmeri* S. Wats.) (Heap 2011). GR *Ambrosia* species are a serious concern because they create greater weed control challenges, are competitive with crops, and cause large crop yield losses. For example, Johnson et al. (2007) observed up to 19% corn yield loss at giant ragweed densities of 0.5 plants m⁻² under season-long interference. Furthermore, Harrison et al. (2001) observed that corn yield loss could be predicted up to 90% for densities of 1.4 plants m⁻², and Baysinger and Sims (1991) observed nearly complete soybean

Table 3. List of the 10 most problematic weeds in the Eastern Corn Belt, United States, from 1990 to 2005.

Rank	1990	1996	2000	2003	2005
1	Canada thistle (<i>Cirsium arvense</i>)	Giant ragweed	Giant ragweed	Giant ragweed	Giant ragweed
2	Foxtail species (<i>Setaria</i> spp.)	Canada thistle	Canada thistle	Common lambsquarters	Johnsongrass
3	Velvetleaf (<i>Abutilon theophrasti</i>)	Hemp dogbane (<i>Apocynum cannabinum</i>)	Johnsongrass (<i>Sorghum halepense</i>)	Canada thistle	Foxtail species (<i>Setaria</i> spp.)
4	Common ragweed	Common lambsquarters	Common lambsquarters	Common cocklebur	Burcucumber
5	Common cocklebur (<i>Xanthium strumarium</i>)	Horseweed	Shattercane	Velvetleaf	Dandelion
6	Giant ragweed	Johnsongrass (<i>Sorghum halepense</i>)	Hemp dogbane	Horseweed	Waterhemp species (<i>Amaranthus</i> spp.)
7	Morningglory species (<i>Ipomoea</i> spp.)	Burcucumber (<i>Sicyos angulatus</i>)	Burcucumber	Waterhemp species (<i>Amaranthus</i> spp.)	Horseweed
8	Quackgrass	Shattercane	Velvetleaf	Burcucumber	Velvetleaf
9	Common lambsquarters (<i>Chenopodium album</i>)	Giant foxtail	Common ragweed	Chickweed (<i>Caryophyllaceae</i> spp.)	Common lambsquarters
10	Pennsylvania smartweed (<i>Polygonum pennsylvanicum</i>)	Fall panicum	Common cocklebur	Dandelion (<i>Taraxacum officinale</i>)	Common cocklebur
Source	Loux and Berry 1991	Childs et al. 1997	Nice and Johnson 2005	Nice and Johnson 2005	Kruger et al. 2008

yield loss from season-long competition of 22 and 36 giant ragweed plants m^{-2} .

GR common waterhemp biotypes have also evolved in GR soybean fields in seven states (Heap 2011). Furthermore, the Missouri GR population is also resistant to ALS- and protoporphyrinogen oxidase (PPO)-inhibiting herbicides (Legleiter and Bradley 2008), and the Illinois population of GR common waterhemp is also multiple resistant with ALS-inhibiting herbicides (Patzoldt et al. 2005) (Heap 2011). In the southern and southeastern United States, GR Palmer amaranth is a competitive weed in GR cotton (Culpepper and York 2008). Much like giant ragweed in corn and soybean production, Palmer amaranth is highly competitive with cotton and soybean, causing potential crop yield losses up to 100% (Culpepper et al. 2006). GR Palmer amaranth and common waterhemp are additionally problematic, especially in geographies these species share, because they have a high potential for introgression of resistance traits due to their dioecious biology and reproduction (Steckel 2007; Trucco et al. 2007; Wetzel et al. 1999). Glyphosate resistance in *Amaranthus* species will be detrimental to crop yields and profitability if alternative approaches or herbicides, such as the auxinic herbicides, are not adopted by producers to slow the selection for GR weed populations.

Herbicide Rotations and Tank Mixes in Auxinic Herbicide-Resistant Crops. Rotations and mixtures of herbicides that have discrete sites of action and are degraded by a common metabolic pathway have been proposed as means for preventing or delaying resistance evolution in weed populations (Beckie and Reboud 2009; Gressel and Segel 1990; Powles et al. 1997; Wrubel and Gressel 1994). Powles et al. (1997) modeled herbicide resistance evolution in a weed population of infinite size when two herbicides were rotated annually or used each year as a mixture. In the absence of fitness penalties, herbicide rotation did not increase the number of applications before resistance was detected for either herbicide. When the herbicides were used in tank mixtures, resistance was delayed by approximately 4 yr. These results were supported by Diggle et al. (2003), who modeled development of herbicide-resistant weed populations and found that the probability of developing resistance to one or both herbicides in a tank mix decreases as the size of the area/initial population decreases. With specific parameters of treatment areas of 100 ha^2 or less, an initial weed seedbank of 100 seeds m^2 and initial frequencies of the resistance genes of 10^6 , development of resistance to both herbicides is not likely within 50 yr for all types of weeds if both herbicides are used every year in tank mixtures. If herbicides are used in alternate years (i.e., in rotation) then multiple resistance almost always occurs in 100 ha^2 areas, but is uncommon in areas of 1 ha^2 or less. Their results also suggest that adoption of practices to limit the movement of weed propagules, in conjunction with using herbicides in combination rather than in rotation, can substantially delay the evolution of herbicide resistance in weeds.

Currently, annual broadleaf weeds in genera such as *Ambrosia*, *Amaranthus*, *Chenopodium*, *Ipomoea*, and *Conyza* have become difficult to control for many crop producers in GR cropping systems. It is interesting to note that biotypes from three weed species (kochia, Russian thistle, and common lambsquarters) in the Chenopodiaceae have evolved

resistance to dicamba (Heap 2011; Table 1), and one cultivated crop species (sugar beet; *Beta vulgaris* L.) in this family is naturally tolerant to the pyridine class of auxinic herbicides (Figure 1), suggesting that weeds in this family may have a higher risk for development of resistance to the auxinic herbicides. New technology on the horizon, such as 2,4-D and dicamba-resistant crops, will provide control options for some GR broadleaf weeds and other weeds that are difficult to control with glyphosate. 2,4-D controls giant ragweed and common waterhemp, but has displayed variable control of horseweed (W. G. Johnson, unpublished data). In a recent regional study (Johnson et al. 2010), the use of dicamba POST in dicamba-resistant soybeans improved the uniformity of control for velvetleaf, smooth pigweed, morningglory, and glyphosate-sensitive waterhemp. Additionally, combining 0.28 kg dicamba ha^{-1} with glyphosate resulted in 30 to 65% greater control of GR Palmer amaranth, GR common waterhemp, GR horseweed, and GR giant ragweed compared with sequentially applied glyphosate applications. In the future, it could be predicted that grasses may become more problematic since auxinic herbicides (as well as glufosinate) have weak activity on grasses and auxinic herbicides occasionally antagonize POST grass control. Additionally, increased use of auxinic herbicides in combination with glyphosate in herbicide-resistant crop varieties should offer effective, short-term solutions for control of glyphosate-, ALS-, triazine-, PPO-, or 4-hydroxyphenyl-pyruvate dioxygenase-resistant dicot weeds.

Acknowledgment

We thank Dr. Gerry Stephenson for his helpful insight and comments regarding the manuscript.

Literature Cited

- Abdullah, I., A. J. Fischer, C. L. Elmore, M. E. Saltveit, and M. Zaki. 2006. Mechanism of resistance to quinclorac in smooth crabgrass (*Digitaria ischaemum* L.). *Pestic. Biochem. Physiol.* 84:38–48.
- Ashton, F. M. and A. S. Crafts. 1981. Phenoxys. Pages 272–302 in F. M. Ashton and A. S. Crafts, eds. *Mode of Action of Herbicides*. Toronto: John Wiley and Sons.
- Bajguz, A. and A. Piotrowska. 2009. Conjugates of auxin and cytokinin. *Phytochemistry* 70:957–969.
- Baysinger, J. A. and B. D. Sims. 1991. Giant ragweed (*Ambrosia trifida*) interference in soybeans (*Glycine max*). *Weed Sci.* 39:358–362.
- Beckie, H. J. and X. Reboud. 2009. Selecting for weed resistance: herbicide rotation and mixture. *Weed Technol.* 23:363–370.
- Berversdorf, W. D., D. J. Hume, and M. J. Donnelly-Vanderloo. 1988. Agronomic performance of triazine-resistant and susceptible reciprocal spring canola hybrids. *Crop Sci.* 28:932–934.
- Bourdot, G. W., D. J. Saville, and G. A. Hurrell. 1996. Ecological fitness and the decline of resistance to the herbicide MCPA in a population of *Ranunculus acris*. *J. Appl. Ecol.* 33:151–160.
- Bradshaw, L. D., S. R. Padgett, S. L. Kimball, and B. H. Wells. 1997. Perspectives on glyphosate resistance. *Weed Technol.* 11:189–198.
- Burke, I. C., J. P. Yenish, D. Pittmann, and R. S. Gallagher. 2009. Resistance of a prickly lettuce (*Lactuca serriola*) biotype to 2,4-D. *Weed Technol.* 23:586–591.
- Calderon-Villalobos, L. I., X. Tan, N. Zheng, and M. Estelle. 2010. Auxin perception—structural insights. *Cold Spring Harb. Perspect. Biol.* 2:a005546.
- Chapman, E. J. and M. Estelle. 2009. Mechanism of auxin-regulated gene expression in plants. *Annu. Rev. Genet.* 43:265–285.
- Childs, D. J., T. N. Jordan, and R. L. Blackwell. 1997. Survey of problem weeds in Indiana: 1996. W. Lafayette, IN: Purdue University Cooperative Extension Service, WS-10.

- Coupland, D. 1994. Resistance to auxin analog herbicides. Pages 171–214 in S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants: Biology and Biochemistry*. Boca Raton, FL: Lewis Publishers, CRC Press.
- Cranston, H. J., A. J. Kern, J. L. Hackett, E. K. Miller, B. D. Maxwell, and W. E. Dyer. 2001. Dicamba resistance in kochia. *Weed Sci.* 49:164–170.
- Culpepper, S. A. 2006. Glyphosate-induced weed shifts. *Weed Technol.* 20: 277–281.
- Culpepper, S. A., T. L. Grey, W. K. Vencill, J. M. Kichler, T. M. Webster, S. M. Brown, A. C. York, J. W. Davis, and W. W. Hanna. 2006. Glyphosate-resistant Palmer amaranth (*Amaranthus palmeri*) confirmed in Georgia. *Weed Sci.* 54:620–626.
- Culpepper, S. A. and A. C. York. 2008. Glyphosate-resistant Palmer amaranth impacts Southeastern agriculture. *Proc. IL. Crop Protect. Technol. Conf.* 60:61–63.
- Darmency, H. 1994. Genetics of herbicide resistance in weeds and crops. Pages 263–297 in S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants: Biology and Biochemistry*. Boca Raton, FL: Lewis Publishers, CRC Press.
- Dauer, J. T., D. A. Mortensen, and R. Humston. 2006. Controlled experiments to predict horseweed (*Coryza canadensis*) dispersal distances. *Weed Sci.* 54:484–489.
- Davis, V. M., K. D. Gibson, T. T. Bauman, S. C. Weller, and W. G. Johnson. 2007. Influence of weed management practices and crop rotation on glyphosate-resistant horseweed population dynamics and crop yield. *Weed Sci.* 55:508–516.
- Davis, V. M., K. D. Gibson, and W. G. Johnson. 2008. A field survey to determine distribution and frequency of glyphosate-resistant horseweed (*Coryza canadensis*) in Indiana. *Weed Technol.* 22:331–338.
- Debreuil, D. J., L. F. Friesen, and I. N. Morrison. 1996. Growth and seed return of auxin-type herbicide resistant wild mustard (*Brassica kaber*) in wheat. *Weed Sci.* 44:872–878.
- DeGennaro, F. P. and S. C. Weller. 1984. Differential susceptibility of field bindweed (*Convolvulus arvensis*) biotypes to glyphosate. *Weed Sci.* 32:472–476.
- Devine, M. D., S. O. Duke, and C. Fedtke. 1993. Herbicides with auxin activity. Pages 295–309 in *Physiology of Herbicide Action*. Englewood Cliffs, NJ: Prentice Hall PTR.
- Devine, M. D. and A. Shukla. 2000. Altered target site as a mechanism of herbicide resistance. *Crop Prot.* 19:881–889.
- Dharmasiri, N., S. Dharmasiri, and M. Estelle. 2005. The F-box protein TIR1 is an auxin receptor. *Nature* 435:441–445.
- Diggle, A. J., P. B. Neve, and F. P. Smith. 2003. Herbicides used in combination can reduce the probability of herbicide resistance in finite weed populations. *Weed Res.* 43:371–382.
- Duke, S. O. 1996. Herbicide-resistant crops: background and perspectives. Pages 1–10 in S. O. Duke, ed. *Herbicide-Resistant Crops: Agricultural, Environmental, Economic, Regulatory, and Technical Aspects*. Boca Raton, FL: CRC Press.
- Dyer, W. E., G. A. Goss, and P. Buck. 2002. Gravitropic responses of auxinic herbicide-resistant *Kochia scoparia*. *Annu. Meet. Weed Sci. Soc. Amer. Abstr.* 171. Pg. 49.
- Firbank, L. G., P. Rothery, and M. J. May, et al. 2006. Effects of genetically modified herbicide-tolerant cropping systems on weed seedbanks in two years of following crops. *Biol. Lett.* 2:140–143.
- Fuerst, E. P., T. M. Sterling, M. A. Norman, T. S. Prather, G. P. Irzyk, Y. Wu, N. K. Lownds, and R. H. Callihan. 1996. Physiological characterization of picloram resistance in yellow starthistle. *Pestic. Biochem. Physiol.* 56:149–161.
- Ge, X., D. A. d'Avignon, J.J.H. Ackerman, and R. D. Sammons. 2010. Rapid vacuolar sequestration: the horseweed glyphosate resistance mechanism. *Pest Manag. Sci.* 66:345–348.
- Goss, G. A. and W. E. Dyer. 2003. Physiological characterization of auxinic herbicide-resistant biotypes of kochia (*Kochia scoparia*). *Weed Sci.* 51: 839–844.
- Gressel, J. and G. Ben-Sinai. 1989. Low intraspecific competitive fitness in a triazine resistant, nearly isogenic line of *Brassica napus*. Pages 489–504 in L. van Vloten-Doting, G.S.P. Groot, and T. C. Hall, eds. *Molecular Form and Function of Plant Genome*. New York: Plenum Press.
- Gressel, J. and L. A. Segel. 1982. Interrelating factors controlling the rate of appearance of resistance: the outlook for the future. Pages 325–447 in H. M. LeBaron and J. Gressel, eds. *Herbicide Resistance in Plants*. New York: John Wiley and Sons.
- Gressel, J. and L. A. Segel. 1990. Modelling the effectiveness of herbicide rotations and mixtures as strategies to delay or preclude resistance. *Weed Technol.* 4:186–198.
- Grossman, K. 2000. The mode of action of auxin herbicides: a new ending to a long, drawn out story. *Trends Plant Sci.* 5:506–508.
- Grossman, K. 2010. Auxin herbicides: current status of mechanism and mode of action. *Pest Manag. Sci.* 66:113–120.
- Grossmann, K. and H. Hansen. 2001. Ethylene-triggered abscisic acid: a principle in plant growth regulation? *Physiol. Plant.* 113:9–14.
- Grossmann, K., F. Scheltrup, J. Kwiatkowski, and G. Caspar. 1996. Induction of abscisic acid is a common effect of auxin herbicides in susceptible plants. *J. Plant Physiol.* 149:475–478.
- Guilfoyle, T. 2007. Sticking with auxin. *Nature* 446:621–622.
- Gustafson, D. I. 2008. Sustainable use of glyphosate in North American cropping systems. *Pest Manag. Sci.* 64:409–416.
- Hall, J. C., S.M.M. Alam, and D. P. Murr. 1993. Ethylene biosynthesis following foliar application of picloram to biotypes of wild mustard (*Sinapis arvensis* L.) susceptible or resistant to auxinic herbicides. *Pestic. Biochem. Physiol.* 47:36–43.
- Hall, J. C. and M. L. Romano. 1995. Morphological and physiological differences between the auxinic herbicide susceptible (S) and resistant (R) wild mustard (*Sinapis arvensis*) biotypes. *Pestic. Biochem. Physiol.* 52: 149–155.
- Hall, J. C., S. R. Webb, and S. Deshpande. 1996. An overview of auxinic herbicide resistance: wild mustard as a case study. Pages 28–43 in T. M. Brown, ed. *Molecular Genetics and Evolution of Pesticide Resistance*. Washington, DC: American Chemical Society.
- Hanson, N. S. 1962. Weed control practices and research for sugar cane in Hawaii. *Weeds* 10:192–200.
- Harper, J. L. 1956. The evolution of weeds in relation to resistance to herbicides. *Proc. 3rd Bright. Weed Control Conf.* 3:179–188.
- Harrison, S. K., E. E. Regnier, J. T. Schmol, and J. E. Webb. 2001. Competition and fecundity of giant ragweed in corn. *Weed Sci.* 49:224–229.
- Heap, I. 2011. International Survey of Herbicide-Resistant Weeds. <http://www.weedscience.org/in.asp>. Accessed: April 14, 2011.
- Heap, I. M. and I. N. Morrison. 1992. Resistance to auxin-type herbicides in wild mustard (*Sinapis arvensis* L.) populations in western Canada. *Annu. Meet. Weed Sci. Soc. Amer. Abstr.* 32:164.
- Hilgenfeld, K. L., A. R. Martin, D. A. Mortensen, and S. C. Mason. 2004. Weed management in a glyphosate resistant soybean system: weed species shifts. *Weed Technol.* 18:284–291.
- Industry Task Force II on 2,4-D Research Data. 2005. Issue Background. <http://www.24d.org/background/24D-Background-Benefits.pdf>. Accessed: October 31, 2010.
- Jacob, B. F., J. H. Duesing, J. Antonovics, and D. T. Patterson. 1988. Growth performance of triazine-resistant and -susceptible biotypes of *Solanum nigrum* over a range of temperatures. *Can. J. Bot.* 66:847–850.
- Jasieniuk, M., A. L. Brule-Babel, and I. N. Morrison. 1996. The evolution and genetics of herbicide resistance in weeds. *Weed Sci.* 44:176–193.
- Jasieniuk, M., I. N. Morrison, and A. L. Brule-Babel. 1995. Inheritance of dicamba resistance in wild mustard (*Brassica kaber*). *Weed Sci.* 43:192–195.
- Jeschke, M. R. and D. E. Stoltenberg. 2006. Weed community composition after eight years of continuous glyphosate use in a corn-soybean annual rotation. *Proc. N. Cent. Weed Sci. Soc.* 58:59.
- Johnson, B., B. Young, and J. Matthews, et al. 2010. Weed control in dicamba resistant soybeans. *Crop Manag.* [Online: doi:10.1094/CM-2010-0920-01-RS].
- Johnson, W. G., E. J. Ott, K. D. Gibson, R. L. Nielsen, and T. T. Bauman. 2007. Influence of nitrogen application timing on low-density giant ragweed (*Ambrosia trifida*) interference in corn. *Weed Technol.* 21:763–767.
- Jugulam, M., M. D. McLean, and J. C. Hall. 2005. Inheritance of picloram and 2,4-D resistance in wild mustard (*Brassica kaber*). *Weed Sci.* 53:417–423.
- Kelley, K. B., K. N. Lambert, A. G. Hager, and D. E. Riechers. 2004. Quantitative expression analysis of *GH3*, a gene induced by plant growth regulator herbicides in soybean. *J. Agric. Food Chem.* 52:474–478.
- Kelley, K. B. and D. E. Riechers. 2007. Recent developments in auxin biology and new opportunities for auxinic herbicide research. *Pestic. Biochem. Physiol.* 89:1–11.
- Kepinski, S. and O. Leyser. 2005. The *Arabidopsis* F-box protein TIR1 is an auxin receptor. *Nature* 435:446–451.
- Kern, A. J., M. E. Chaverra, H. J. Cranston, and W. E. Dyer. 2005. Dicamba-responsive genes in herbicide-resistant and susceptible biotypes of kochia (*Kochia scoparia*). *Weed Sci.* 53:139–145.
- Kirby, C. 1980. *The Hormone Weedkillers: A Short History of Their Discovery and Development*. London Road, Croydon, UK: British Crop Protection Council. 55 p.
- Kruger, G. R., W. G. Johnson, S. C. Weller, M.D.K. Owen, D. R. Shaw, J. W. Wilcut, D. L. Jordan, R. G. Wilson, and B. G. Young. 2008. U.S. grower

- views on problematic weeds and changes in weed pressure in glyphosate-resistant corn, cotton, and soybean cropping systems. *Weed Technol.* 23:162–166.
- Lee, O. C. 1948. The effect of 2,4-D as a selective herbicide in growing corn and sorghums. *Proc. North Cent. Weed Sci. Soc.* 5:18–21.
- Legleiter, T. R. and K. W. Bradley. 2008. Glyphosate and multiple herbicide resistance in waterhemp (*Amaranthus rudis*) populations from Missouri. *Weed Sci.* 56:582–587.
- Loux, M. M. and M. A. Berry. 1991. Use of a grower survey for estimating weed problems. *Weed Technol.* 5:460–466.
- Lovelace, M. L., R. E. Talbert, R. E. Hoagland, and E. F. Scherder. 2007. Quinclorac absorption and translocation characteristics in quinclorac- and propanil-resistant and -susceptible barnyardgrass (*Echinochloa crus-galli*) biotypes. *Weed Technol.* 21:683–687.
- Ludwig-Müller, J. 2011. Auxin conjugates: their role for plant development and in the evolution of land plants. *J. Expt. Bot.* 62:1757–1773.
- McCloskey, W. B. and J. S. Holt. 1991. Effect of growth temperature on biomass production of nearly isonuclear triazine-R and S common groundsel (*Senecio vulgaris* L.). *Plant Cell Environ.* 14:699–705.
- Mithila, J. and J. C. Hall. 2005. Comparison of ABP1 over-expressing *Arabidopsis* and under-expressing tobacco with an auxinic herbicide-resistant wild mustard (*Brassica kaber*) biotype. *Plant Sci.* 169:21–28.
- Mockaitis, K. and M. Estelle. 2008. Auxin receptors and plant development: a new signaling paradigm. *Annu. Rev. Cell Dev. Biol.* 24:55–80.
- Morrison, I. N. and M. D. Devine. 1994. Herbicide resistance in the Canadian prairie provinces: five years after the fact. *Phytoprotection* 75(Suppl.):5–16.
- Nice, G. and B. Johnson. 2005. Indiana's Top Ten Most Problematic Weeds. Purdue University Cooperative Extension Services. www.btny.purdue.edu/WeedScience/2005/topten05.pdf. Accessed: October 31, 2010.
- Owen, M.D.K. and I. A. Zelaya. 2005. Herbicide-resistant crops and weed resistance to herbicides. *Pest Manag. Sci.* 61:301–311.
- Patzoldt, W. L., P. J. Tranel, and A. G. Hager. 2005. A waterhemp (*Amaranthus tuberculatus*) biotype with multiple resistances across three herbicide sites of action. *Weed Sci.* 53:30–36.
- Peniuk, M. G., M. L. Romano, and J. C. Hall. 1993. Physiological investigations into the resistance of wild mustard (*Sinapis arvensis* L.) biotype to auxinic herbicides. *Weed Res.* 33:431–440.
- Peterson, S. V., A. I. Johansson, M. Kowalczyk, A. Makoveychuk, J. Y. Wang, T. Moritz, M. Grebe, P. N. Benfey, G. Sandberg, and K. Ljung. 2009. An auxin gradient and maximum in the *Arabidopsis* root apex shown by high-resolution cell-specific analysis of IAA distribution and synthesis. *Plant Cell* 21:1659–1668.
- Powles, S. B. and C. Preston. 2006. Evolved glyphosate resistance in plants: biochemical and genetic basis of resistance. *Weed Technol.* 20:282–289.
- Powles, S. B., C. Preston, I. B. Bryan, and A. R. Jutsum. 1997. Herbicide resistance: impact and management. *Advan. Agron.* 58:57–93.
- Preston, C., D. S. Belles, P. H. Westra, S. J. Nissen, and S. M. Ward. 2009. Inheritance of resistance to the auxinic herbicide dicamba in kochia (*Kochia scoparia*). *Weed Sci.* 57:43–47.
- Preston, C. and C. A. Mallory-Smith. 2001. Biochemical mechanisms, inheritance, and molecular genetics of herbicide resistance in weeds. Pages 23–60 in S. B. Powles and D. L. Shaner, eds. *Herbicide Resistance in World Grains*. Boca Raton, FL: CRC Press.
- Romero-Puertas, M. C., I. McCarthy, M. Gomez, L. M. Sandalio, F. J. Corpas, L. A. Del Rio, and J. M. Palma. 2004. Reactive oxygen species-mediated enzymatic systems involved in the oxidative action of 2,4-dichlorophenoxyacetic acid. *Plant Cell Environ.* 27:1135–1148.
- Rubery, P. H. 1977. The specificity of carrier-mediated auxin transport by suspension-cultured crown gall cells. *Planta* 135:275–283.
- Sabba, R. P., I. M. Ray, N. Lownds, and T. M. Sterling. 2003. Inheritance of resistance to clopyralid and picloram in yellow starthistle (*Centaurea solstitialis*) is controlled by a single nuclear recessive gene. *J. Hered.* 94:523–527.
- Sabba, R. P., T. M. Sterling, and N. Lownds. 1998. Effect of picloram on R and S yellow starthistle (*Centaurea solstitialis*): the role of ethylene. *Weed Sci.* 46:297–300.
- Salisbury, F. B. and C. W. Ross. 1992. *Plant Physiology*. 4th ed. Belmont, CA: Wadsworth Publishing Company. 635 p.
- Shaner, D. L. 2000. The impact of glyphosate-tolerant crops on the use of other herbicides and on resistance management. *Pest Manag. Sci.* 56:320–326.
- Simon, S. and J. Petrášek. 2011. Why plants need more than one type of auxin. *Plant Sci.* 180:454–460.
- Singh, D. 2009. Understanding 2,4-D resistance in prickly lettuce (*Lactuca serriola*) and evaluating chemical fallow systems for the inland PNW. Ph.D. dissertation. Pullman, WA: Washington State University. 159 p.
- Staswick, P. E. 2009. The tryptophan conjugates of jasmonic and indole-3-acetic acids are endogenous auxin inhibitors. *Plant Physiol.* 150:1310–1321.
- Staswick, P. E., B. Serban, M. Rowe, I. Tiryaki, M. T. Maldonado, M. C. Maldonado, and W. Suza. 2005. Characterization of an *Arabidopsis* enzyme family that conjugates amino acids to indole-3-acetic acid. *Plant Cell* 17:616–627.
- Steckel, L. E. 2007. The dioecious *Amaranthus* spp.: here to stay. *Weed Technol.* 21:567–570.
- Sterling, T. M. 1994. Mechanisms of herbicide absorption across plant membranes and accumulation in plant cells. *Weed Sci.* 42:263–276.
- Sterling, T. M. and J. C. Hall. 1997. Mechanism of action of natural auxins and the auxinic herbicides. Pages 111–141 in R. M. Roe, J. D. Burton, and R. J. Kuhr, eds. *Herbicide Activity: Toxicology, Biochemistry and Molecular Biology*. Amsterdam, the Netherlands: IOS Press.
- Tan, X., L.I.A. Calderon-Villalobos, M. Sharon, C. Zheng, C. V. Robinson, M. Estelle, and N. Zheng. 2007. Mechanism of auxin perception by the TIR1 ubiquitin ligase. *Nature* 446:640–645.
- Triplett, G. B., Jr. and G. D. Lytle. 1972. Control and ecology of weeds in continuous corn grown without tillage. *Weed Sci.* 20:453–457.
- Tromas, A., I. Paponov, and C. Perrot-Rechenmann. 2010. Auxin binding protein 1: functional and evolutionary aspects. *Trends Plant Sci.* 15:436–446.
- Trucco, F., D. Zheng, A. J. Woodyard, J. R. Walter, T. C. Tatum, A. L. Rayburn, and P. J. Tranel. 2007. Nonhybrid progeny from crosses of dioecious amaranths: implications for gene-flow research. *Weed Sci.* 55: 119–122.
- United States Department of Agriculture - National Agricultural Statistics Service. USDA-NASS. 2008. Agricultural Chemical Use Database. www.pestmanagement.info/nass. Washington, DC: USDA-NASS. Accessed: October 31, 2010.
- Valenzuela-Valenzuela, J. M., N. K. Lownds, and T. M. Sterling. 2001. Clopyralid uptake, translocation, metabolism, and ethylene induction in picloram-resistant yellow starthistle (*Centaurea solstitialis* L.). *Pestic. Biochem. Physiol.* 71:11–19.
- Valenzuela-Valenzuela, J. M., N. K. Lownds, and T. M. Sterling. 2002. Ethylene plays no role in clopyralid action in yellow starthistle (*Centaurea solstitialis* L.). *Pestic. Biochem. Physiol.* 72:142–152.
- Van Eerd, L. L., M. D. McLean, G. R. Stephenson, and J. C. Hall. 2004. Resistance to quinclorac and ALS-inhibitor herbicides in *Galium spurium* is conferred by two distinct genes. *Weed Res.* 44:355–365.
- Van Eerd, L. L., G. R. Stephenson, J. Kwiatkowski, K. Grossmann, and J. C. Hall. 2005. Physiological and biochemical characterization of quinclorac and resistance in a false cleavers (*Galium spurium*) biotype. *J. Agric. Food Chem.* 53:1144–1151.
- VanGessel, M. J. 2001. Glyphosate resistant horseweed from Delaware. *Weed Sci.* 49:703–705.
- Vila-Aiub, M. M., P. Neve, and S. B. Powles. 2009. Fitness costs associated with evolved herbicide resistance alleles in plants. *New Phytol.* 184:751–767.
- Walsh, M. J., N. Maguire, and S. B. Powles. 2009. Combined effects of wheat competition and 2,4-D amine on phenoxy herbicide resistant *Raphanus raphanistrum* populations. *Weed Res.* 49:316–325.
- Walsh, M. J., S. B. Powles, B. R. Beard, B. T. Parkin, and S. A. Porter. 2004. Multiple-herbicide resistance across four modes of action in wild radish (*Raphanus raphanistrum*). *Weed Sci.* 52:8–13.
- Walsh, T. A., R. Neal, A. O. Merlo, M. Honma, G. R. Hicks, K. Wolff, W. Matsumura, and J. P. Davies. 2006. Mutations in an auxin receptor homolog AFB5 and in SGT1b confer resistance to synthetic picolinate auxins and not to 2,4-dichlorophenoxyacetic acid or indole-3-acetic acid in *Arabidopsis*. *Plant Physiol.* 142:542–552.
- Webb, S. R. and J. C. Hall. 1995. Auxinic herbicide-resistant and -susceptible wild mustard (*Sinapis arvensis* L.) biotypes: effect of auxinic herbicides on seedling growth and auxin-binding activity. *Pestic. Biochem. Physiol.* 52:137–148.
- Wei, Y. D., H. G. Zheng, and J. C. Hall. 2000. Role of auxinic herbicide-induced ethylene on hypocotyl elongation and root/hypocotyl radial expansion. *Pest Manag. Sci.* 56:377–387.
- Weinberg, T., G. R. Stephenson, M. D. McLean, and J. C. Hall. 2006. MCPA (4-chloro-2-ethylphenoxyacetate) resistance in hemp-nettle (*Galeopsis tetrahit* L.). *J. Agric. Food Chem.* 54:9126–9134.
- Went, F. W. 1926. Growth accelerating substances in coleoptile of *Avena sativa*. *Proc. K. Akad. Wet. Amsterdam* 30:10–19.
- Wetzel, D. K., M. J. Horak, D. Z. Skinner, and P. A. Kulakow. 1999. Transferal of herbicide resistance traits from *Amaranthus palmeri* to *Amaranthus rudis*. *Weed Sci.* 47:538–543.
- Wilson, R. G., S. D. Miller, P. Westra, A. R. Kniss, P. W. Stahlman, G. W. Wicks, and S. D. Kachman. 2007. Glyphosate-induced weed shifts in glyphosate-resistant corn or a rotation of glyphosate-resistant corn, sugarbeet, and spring wheat. *Weed Technol.* 21:900–909.

Wrubel, R. P. and J. Gressel. 1994. Are herbicide mixtures useful for delaying the rapid evolution of resistance? A case study. *Weed Technol.* 8:635–648.

Zhang, Q. and D. E. Riechers. 2008. Proteomics: an emerging technology for weed science research. *Weed Sci.* 56:306–313.

Zheng, H. G. and J. C. Hall. 2001. Understanding auxinic herbicide resistance in *Sinapis arvensis* L.: physiological, biochemical and molecular genetic approaches. *Weed Sci.* 49:276–281.

Received April 24, 2011, and approved April 27, 2011.