

Herbicide-Resistant Weeds: Management Tactics and Practices

Author(s): HUGH J. BECKIE

Source: Weed Technology, 20(3):793-814. 2006.

Published By: Weed Science Society of America

DOI: <http://dx.doi.org/10.1614/WT-05-084R1.1>

URL: <http://www.bioone.org/doi/full/10.1614/WT-05-084R1.1>

BioOne (www.bioone.org) is a nonprofit, online aggregation of core research in the biological, ecological, and environmental sciences. BioOne provides a sustainable online platform for over 170 journals and books published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Web site, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/page/terms_of_use.

Usage of BioOne content is strictly limited to personal, educational, and non-commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

Herbicide-Resistant Weeds: Management Tactics and Practices¹

HUGH J. BECKIE²

Abstract: In input-intensive cropping systems around the world, farmers rarely proactively manage weeds to prevent or delay the selection for herbicide resistance. Farmers usually increase the adoption of integrated weed management practices only after herbicide resistance has evolved, although herbicides continue to be the dominant method of weed control. Intergroup herbicide resistance in various weed species has been the main impetus for changes in management practices and adoption of cropping systems that reduce selection for resistance. The effectiveness and adoption of herbicide and nonherbicide tactics and practices for the proactive and reactive management of herbicide-resistant (HR) weeds are reviewed. Herbicide tactics include sequences and rotations, mixtures, application rates, site-specific application, and use of HR crops. Nonherbicide weed-management practices or nonselective herbicides applied preplant or in crop, integrated with less-frequent selective herbicide use in diversified cropping systems, have mitigated the evolution, spread, and economic impact of HR weeds.

Additional index words: Herbicide resistance, integrated weed management.

Abbreviations: ACCase, acetyl-CoA carboxylase; ALS, acetolactate synthase; APP, aryloxyphenoxypionate; CHD, cyclohexanedione; DSS, decision-support system; EPSPS, enolpyruvylshikimate-3-phosphate synthase; HR, herbicide resistant; HS, herbicide susceptible; IWM, integrated weed management.

INTRODUCTION

The main risk factors for the evolution of HR weeds are: (a) recurrent application of highly efficacious herbicides with the same site of action; (b) annual weed species that occur at high population densities, are widely distributed, genetically variable, prolific seed producers, and have efficient gene (seed or pollen) dissemination; and (c) simple cropping systems that favor a few dominant weed species (Owen 2001a; Thill and Lemerle 2001). Globally, the most economically important HR weeds include rigid (annual) ryegrass (*Lolium rigidum* Gaudin), wild oat (*Avena fatua* L.), *Amaranthus* spp. (redroot pigweed, *A. retroflexus* L.; smooth pigweed, *A. hybridus* L.; common waterhemp, *A. rudis* Sauer; and tall waterhemp, *A. tuberculatus* (Moq.) J.D. Sauer), common lambsquarters (*Chenopodium album* L.), green foxtail [*Setaria viridis* (L.) Beauv.], barnyardgrass [*Echinochloa crus-galli* (L.) Beauv.], goosegrass [*Eleusine indica* (L.) Gaertn.], kochia [*Kochia scoparia* (L.)

Schrad.], horseweed [*Conyza canadensis* (L.) Cronq.], and blackgrass (*Alopecurus myosuroides* Huds.) (Heap 2005). For the majority of HR weed biotypes, herbicide resistance is target-site based, conferred by a single, major (i.e., large phenotypic effect) gene with a high degree of dominance (Powles et al. 1997). This mode of inheritance favors the rapid evolution of weed resistance to herbicides applied at registered rates.

In high-input cropping systems around the world, farmers are reluctant to proactively manage weeds to prevent or delay the selection for herbicide resistance. The cost and effort of preventing/delaying resistance to many herbicides are widely perceived or estimated to be the same as that of managing HR weeds, and therefore farmers often do not change their weed management program until resistance has occurred. The lack of proactive management of the evolution of HR weed populations may be due to farmers' primary interest in optimizing short-term economic returns, or inability to assess the economic risks associated with HR weeds (Rotteveel et al. 1997).

Low adoption of resistance-avoidance tactics may also be due to the lack of alternative herbicide groups (defined by site of action) to control the target weeds, or

¹ Received for publication June 14, 2005, and in revised form December 13, 2005.

² Plant Scientist, Agriculture and Agri-Food Canada, Saskatoon Research Centre, 107 Science Place, Saskatoon, Saskatchewan, Canada S7N 0X2. E-mail: beckieh@agr.gc.ca.

Table 1. Use of integrated weed management practices by Western Australian grain farmers in 2000 ($n = 132$) (adapted from Llewellyn et al. 2004).

Practice ^a	Farmer adoption			Expected efficacy ^c
	HR ^b	no HR	All	
	%			
Stubble burning	85	66	76	47 ± 19
Weed seed catching	10	2	7	57 ± 15
Tillage	49	49	46	39 ± 22/49 ± 21
Autumn tickle (preplant tillage)	57	26	44	
Delayed planting	53	35	46	55 ± 23
Double knock	62	49	57	64 ± 21
Crop topping	44	11	30	62 ± 17
Green manuring	21	11	17	74 ± 19
Crop cut for hay	31	49	39	
Spray topping	95	94	94	
High wheat seeding rate	57	54	56	28 ± 17/35 ± 19
Trifluralin ^d				67 ± 14

^a Double knock, crop topping, and spray topping are the use of a nonselective herbicide applied: preplant (followed by another nonselective herbicide or tillage), to annual legumes at postanthesis weed growth stage, and to pastures, respectively, to reduce weed seed production.

^b HR: farmers with herbicide resistance ($n = 77$) vs. no resistance ($n = 55$).

^c Efficacy expected by farmers for rigid ryegrass (*Lolium rigidum* Gaudin) control ± standard deviation; tillage, high seeding rate: difference among nonusers and users, respectively.

^d Included for comparison.

unrealistic expectations that new herbicide technology will continually be forthcoming (Llewellyn et al. 2002). However, herbicides should be viewed as a nonrenewable resource. With the cost of discovering, developing, and marketing a novel herbicide at approximately United States (U.S.) \$150 to \$180 million in 2005 (D. Porter, personal communication), farmers cannot expect many compounds with novel sites of action to be commercialized in the near future.

Additionally, a lack of information on the impact of management tactics and practices on selection of herbicide resistance may limit a farmer's ability to delay resistance. How long herbicide resistance can be delayed by implementing a comprehensive integrated weed-management (IWM) program is uncertain. Moreover, recommendations to farmers to delay or prevent herbicide resistance are often similar to those recommended for managing resistance, thus discouraging the adoption of prevention tactics. Socioeconomic factors in developed countries, such as farmer demographics, increasing size of farms with concomitant limited labor and time availability, high percentage of leased land by renters with a general lack of awareness of previous herbicide history or reduced motivation for long-term stewardship, and preference for annual cropping systems based on lifestyle choice and cash flow, reinforces a heavy reliance on herbicides as the dominant method of weed control (Friesen et al. 2000).

Farmers usually increase the adoption of IWM practices only after herbicide resistance has evolved (Beckie and Gill, 2006). Populations of a number of HR grass

weed biotypes threaten cereal grain production in different areas of the world. Cross-resistance (a single resistance mechanism conferred by one or more genes) and multiple resistance (two or more resistance mechanisms) in weed species have often been the main impetus for the utilization of a greater number of IWM practices in cropping systems (Powles et al. 1997, 2000). For example, farmers in Western Australia with infestations of HR rigid ryegrass practice weed seed catching at harvest more frequently than those with no resistance (Table 1). Farmers with resistance used an average of 8.4 IWM practices, significantly more than farmers with no resistance (mean of 6.6) (Llewellyn et al. 2004).

Prevention can cost significantly less than dealing with resistance once it fully develops, where intergroup herbicide resistance occurs, or where few alternative herbicides are available (Orson 1999). The greatest direct cost of herbicide resistance to the farmer can occur during the first year of poor weed control and consequent yield loss (Peterson 1999). Populations of weeds with high fecundity potential, such as rigid ryegrass, can increase rapidly after control failures caused by resistance. To manage resistance, farmers first use alternative herbicides (i.e., addition of a tank-mix partner or rotating to a herbicide with a different site of action). In some situations, herbicides that selected for resistance may continue to be used because of their cost-effective (i.e., economical) control of non-HR weed species (e.g., triazines or glyphosate applied to land with triazine- or glyphosate-HR biotypes, respectively). The addition of a herbicide to control the HR weed biotype, however, will

increase costs to the farmer (Peterson 1999). The short-term cost of resistance is minimal if alternative herbicides are available, such as those for control of many biotypes resistant to phenoxy or photosystem I-disrupting herbicides (Beckie et al. 2001b). In contrast, there may be a limited number of herbicide options for control of some intergroup-HR biotypes, and those that are available usually increase costs. For example, most or all alternative herbicides to control intergroup-HR biotypes of wild oat or green foxtail in the northern Great Plains increase costs to farmers (Beckie et al. 1999b, 1999c). Management of glyphosate-HR horseweed in conservation-tillage systems in the North Delta region of the United States requires a phenoxy herbicide and one or two residual herbicides. As a consequence of this additional herbicide cost (U.S. \$16 to \$62/ha), conservation tillage has dropped by about 50% in cotton (*Gossypium hirsutum* L.) and 30% overall (Steckel et al. 2005).

The prime strategy for managing herbicide resistance in weeds is to reduce the selection pressure for resistance evolution by any one selecting agent, while maintaining adequate weed control. Selection pressure has the greatest impact on herbicide-resistance evolution and is a factor that farmers can control. Selection pressure imposed by a herbicide is the product of selection intensity (efficacy) and selection duration (persistence in soil) (Putwain 1982). Herbicides applied in crop generally result in the greatest selection pressure compared with other application timings. Selection pressure against a weed population over time, resulting in increasing frequency of HR individuals that collectively possess one or more resistance mechanisms, is a function of frequency of application. Mathematically, the relative selection pressure of a herbicide on a target weed species in a population has been defined as the proportion of HR plants divided by the proportion of herbicide-susceptible (HS) plants that remain after exposure to the herbicide (Gressel and Segel 1982). These proportions are equal to one minus the effective kill, defined by seed yield reduction (Beckie and Morrison 1993; Gressel and Segel 1982). For example, if seed production of HR and HS biotypes is reduced by 42 and 99%, respectively, relative selection pressure is estimated to be $(1 - 0.42)/(1 - 0.99) = (0.58)/(0.01) = 58$. By definition, the selection pressure can only be reduced by lowering the effective kill of HS plants or increasing the effective kill of HR plants. No selection pressure is exerted when HS and HR genotypes are controlled equally. Diversification of selection pressures on weed populations, such as varying the type and

timing of herbicide application (e.g., selective or non-selective herbicides applied preplant, in crop, preharvest, or postharvest), integrating cultural or mechanical weed management practices with reduced herbicide use, and diversifying the cropping system as a whole, is required to reduce the selection pressure of any one selecting agent (Boerboom 1999).

In this review, tactics and practices to effectively delay or manage HR weeds in input-intensive cropping systems worldwide are summarized. Herbicide-based tactics are emphasized, because herbicides will continue to be the dominant weed-control tool in these cropping systems during the foreseeable future. Nonherbicide tactics and practices that have been proven effective in managing HR weeds are outlined. Examples are provided to illustrate the impact of herbicide and nonherbicide tactics on the successful proactive and reactive management of HR weeds.

HERBICIDE TACTICS

A herbicide sequence is defined as two or more applications of herbicides with different sites of action within one crop, whereas herbicide rotation is the application of herbicides with different sites of action to multiple crops over multiple growing seasons in a field. Herbicide sequences, rotations, or mixtures generally have the greatest effect in delaying resistance when the mechanism conferring resistance is target-site based, the target weed species are highly self-pollinated, and seed spread is restricted (Beckie et al. 2001b; Wrubel and Gressel 1994). Multiple resistance can evolve within a weed population through a change in selection history (usually sequential selection), through selection of multiple mechanisms by a single herbicide, or through outcrossing among individuals containing different resistance mechanisms (Hall et al. 1994; Preston and Mallory-Smith 2001). Based on a compounded resistance frequency model, the probability of HR mutants with multiple mechanisms of resistance (target-site based) in an unselected population is the product of the probabilities of resistance to each affected herbicide site of action and thus is rare (Wrubel and Gressel 1994). However, frequent use of herbicides in a field over time can enrich HR populations with different resistance mechanisms. Outcrossing among plants, such as *Lolium* spp. or black-grass, in close proximity that possess different HR mechanisms can result in multiple-HR progeny. Spreading HR seed within and among fields can also aid this process.

Herbicide Sequences and Rotations. *Adoption.* Performance and cost of herbicides usually rank higher than site of action when farmers select a herbicide. The lack of suitable herbicide options associated with crop rotation can be an impediment to herbicide group rotation (Bourgeois et al. 1997b; Légère et al. 2000). The level of adoption of herbicide group rotation for resistance management has increased markedly during the past decade in Canada and Australia. There is little information on the adoption of this tactic in other countries. In western Canada in 1998, fewer than 50% of farmers practiced herbicide group rotation, even though awareness was high (Beckie et al. 1999a). By 2003, 70 (Saskatchewan) to 90% (Manitoba) of farmers claimed to rotate herbicides by site of action (H. Beckie, unpublished data). In 2005, over half of the herbicide products sold in Canada had resistance management labeling, which includes group identification symbols on the label and guidelines for resistance management tactics in the use directions (N. Malik, personal communication). The guidelines were a joint effort between the Pest Management Regulatory Agency (PMRA 1999) in Canada and the U.S. Environmental Protection Agency (2001). By 1998 in Australia, the adoption rate of herbicide group rotation was 85%, attributed largely to site-of-action labeling on herbicide containers (Shaner et al. 1999). It is the most common herbicide resistance management tactic cited by farmers in survey questionnaires conducted in Australia (Shaner et al. 1999) and Canada (H. Beckie, unpublished data). A prerequisite for herbicide group rotation is keeping field records of herbicides used each year. Software packages of crop and herbicide rotation planners are available in many jurisdictions, which facilitate record keeping and can flag high-risk herbicide practices, such as repeated use of herbicides with the same site of action.

Mitigating herbicide resistance risk. Evolution of target-site resistance in weed biotypes is attributed to frequent use of herbicides of the same site of action and their propensity to select for HR biotypes (Beckie et al. 2001a; LeBaron and McFarland 1990). Knowledge of resistance risk could be an incentive for farmers to practice herbicide sequences or rotations to delay the rate of evolution of resistance. The ease of selection for HR biotypes is governed by several factors. As described previously, the selection pressure (efficacy and persistence) imposed on the target weed species by a herbicide is the most important factor affecting the rate of evolution of resistance. The slow evolution of resistance in weed biotypes to phenoxy herbicides, first introduced in 1946, has been partially attributed to relatively low se-

lection pressure (Coupland 1994). Similarly, relatively low efficacy of trifluralin, a dinitroaniline herbicide, on rigid ryegrass has been cited as one reason for relatively slow evolution of resistance (Table 1). In a 1998 Western Australian field survey of rigid ryegrass, population densities were unrelated to herbicide resistance, suggesting the availability of alternative herbicides, particularly trifluralin, to control HR rigid ryegrass (Llewellyn and Powles 2001).

Nonpersistent herbicides generally exert less selection pressure than those that control successive flushes of germinating weeds throughout the growing season. The contribution of persistence to selection pressure, however, depends on timing of herbicide application and the germination characteristics of the target species in a geographic region. The soil residual activity of herbicides did not strongly influence selection pressure on wild oat in a competitive crop (canola, *Brassica napus* L.) in western Canada (Beckie and Holm 2002). The selection pressure exerted on wild oat by residual herbicides was the same as or lower than that of nonresidual herbicides. In the relatively short growing season in the northern Great Plains, few wild oat plants may emerge after post-emergence application of a nonresidual herbicide and produce viable seeds in a competitive crop. In other agroecoregions, herbicide persistence in soil can have a much greater effect on selection pressure.

Whereas a single mutation can confer resistance to single site-of-action herbicides, multiple mutations within a plant are often needed to confer resistance to herbicides with more than one site of action, such as chloracetamide herbicides (Foes et al. 1998). As indicated previously, individuals in an unselected population with multiple mutations for resistance generally would be rare (Preston and Mallory-Smith 2001; Wrubel and Gressel 1994). The frequency of HR alleles in unselected populations defines the starting point for resistance evolution, and thus impacts the length of time for resistance to evolve to noticeable levels. An unusually low rate of mutation of the locus conferring resistance, or alternatively few fit mutations, are speculated to contribute to the slow evolution of resistance to phenoxy and other herbicides, such as glyphosate (Gressel 1999; Jasieniuk et al. 1995). Fit mutations are more probable for non-competitive inhibitors of target-site enzymes such as acetyl-CoA carboxylase (ACCase, EC 6.4.1.2) and acetolactate synthase (ALS, EC 4.1.3.18), where the herbicide binding site is different from the active site. The probability of finding an initial HR mutant in an unselected population increases with an increase in the number of

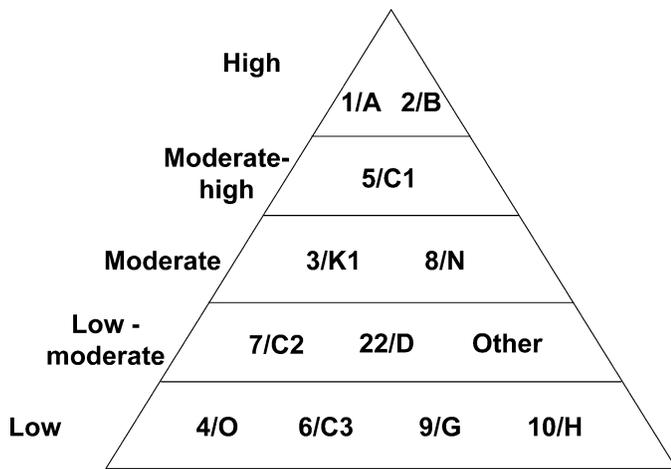


Figure 1. Classification of herbicide site of action by risk of selection for target-site resistance (high ≤ 10 ; moderate = 11–20; low >20 applications (H. Beckie and L. Hall, unpublished data); “Other”: insufficient information to definitively categorize as low or moderate risk. Numerical (Weed Science Society of America) and alphabetical (Herbicide Resistance Action Committee) herbicide groups are described in Mallory-Smith and Retzinger (2003) or Heap (2005).

types of functional mutations (Murray et al. 1996). There are at least five different point mutations in each of the ACCase and ALS target sites in HR weed biotypes, each conferring a different cross-resistance pattern and level of resistance (Délye and Michel 2005; Gressel 2002). Indeed, the frequency of target-site-based ALS inhibitor-HR individuals in untreated populations of rigid ryegrass was found to be relatively high, ranging from 10^{-5} to 10^{-4} (Preston and Powles 2002a). In contrast, most mutations conferring resistance to glyphosate, a competitive inhibitor of enolpyruvylshikimate-3-phosphate synthase (EPSPS, EC 2.5.1.19), and glufosinate, a competitive inhibitor of glutamine synthetase (EC 2.7.7.42), are believed to be lethal.

The risk of target-site resistance, defined by the mean number of applications before resistance is detected, varies by herbicide group (Figure 1). This approach of risk assessment assumes that a particular herbicide site of action is effective for a set number of applications before the onset of target-site resistance. The use of herbicide “shots” is appropriate in economic models and farm-management decision aids (Diggle and Neve 2001). Anecdotal information, namely, field histories of herbicide use, usually is used for assessing the risk of selecting for resistance based on an herbicide’s site of action. Only one long-term experiment has examined the effect of frequency of herbicide use on the evolution of resistance. In a large-plot field experiment conducted from 1979 to 1998, resistance in wild oat to triallate occurred after 18 yr where the herbicide was applied annually in

continuous spring wheat (*Triticum aestivum* L.), but not where it was applied 10 times in a wheat–fallow rotation over the same period (Beckie and Jana 2000).

It is widely agreed that ACCase and ALS inhibitor herbicides pose a high risk for selecting HR biotypes relative to herbicides from other groups (Dellow et al. 1997; Gressel 1997; Heap 1999; LeBaron and McFarland 1990; Monjardino et al. 2003). High-risk herbicides should be applied less often in sequences or rotations than lower-risk herbicides. At a minimum, use of high-risk herbicides in consecutive years in a field should be avoided. In sequences, lower-risk, nonselective herbicides, such as photosystem-I electron diverters (paraquat, diquat) or EPSPS inhibitor (glyphosate) should be used preplant to reduce the number of weeds selected with in-crop herbicides that pose a higher risk. Ideally, high-risk herbicides should not be used in fields with high weed densities, because the number of HR mutants is proportional to population size (Jasieniuk et al. 1996).

Nonselective herbicides, such as paraquat, are commonly applied at the postanthesis stage of rigid ryegrass in annual legume (pulse) crops in Australia, referred to as “crop topping.” In Western Australia, four times as many farmers with HR rigid ryegrass practice crop topping than those with no resistance (Table 1). This practice can markedly reduce weed seed production (Gill and Holmes 1997). In Australia, there has been wide adoption of herbicide techniques to reduce seed production to manage resistance in rigid ryegrass in both annual legume crops and pastures (“spray topping”) (Table 1). The Australian National Glyphosate Sustainability Working Group (2005) recommends reducing the risk of glyphosate resistance by rotating glyphosate with paraquat for preplant weed control, or using a “double knock” (or “double knockdown”) technique by following in sequence a preplant glyphosate application with tillage or a paraquat-based product (Weersink et al. 2005) (Table 1).

Trends discerned in the cross-resistance patterns of weed species resistant to herbicides of the same site of action may be used as a guide for strategic herbicide use. Patterns of cross-resistance, however, cannot be accurately predicted based on field histories of herbicide use. Incidence of aryloxyphenoxypropionate (APP) resistance in HR *Avena* spp. biotypes tends to be greater than that of cyclohexanedione (CHD) resistance in many countries (Beckie et al. 1999b, 1999c, 2002; Cocker et al. 2000; Mansooji et al. 1992; Seefeldt et al. 1994). Thus, as a short-term tactic to manage ACCase target-

site resistance in populations of this species, CHDs may have a higher probability of success. An apparently widespread point mutation in the ACCase gene resulting in an amino-acid change from isoleucine to leucine at position 1781 confers resistance to some APP and CHD herbicides in several grass weed species (Délye et al. 2003; Kaundun and Windass 2004). In various grass weed species, clethodim has often controlled ACCase inhibitor–HR biotypes in dicot crops (Bradley and Hagoood 2001). Apparently, the point mutation(s) that confer(s) resistance to this herbicide occurs relatively infrequently.

Individuals in a population exposed to the same selection pressure can exhibit different patterns of cross-resistance, however, highlighting the probable short-term success of this approach. Wild oat patches with different cross-resistance patterns have been documented within a field (Andrews et al. 1998; Bourgeois et al. 1997a). ALS inhibitor resistance in a population of prostrate pigweed (*Amaranthus blitoides* S. Wats.) in a field in Israel is endowed by two point mutations, each conferring a different cross-resistance pattern (Sibony and Rubin 2003). Allele-specific assays can detect different point mutations (Délye et al. 2002; Kaundun and Windass 2004; Siminszky et al. 2005). Such assays are being commercialized to determine cross-resistance patterns rapidly in HR weed populations where resistance is target-site based, providing farmers with the option of applying an effective herbicide within the same growing season as weed tissue samples are collected for testing.

Herbicide resistance is often attributed to a lack of herbicide group rotation, that is, frequent or repeated use of herbicides of the same site of action. However, there is direct epidemiological evidence for the utility of herbicide group rotations in delaying the evolution of target-site resistance. Examples where herbicide group rotation has been credited in preventing or delaying resistance in weeds include triazine-HR weeds in North America (Stephenson et al. 1990), isoproturon-HR littleseed canarygrass (*Phalaris minor* Retz.) in India (Singh et al. 1999), ACCase inhibitor–HR wild oat in Canada (Légère et al. 2000) and rigid ryegrass in Australia (Gill 1995), and ALS inhibitor–HR common cocklebur (*Xanthium strumarium* L.) in the southern U.S. (Schmidt et al. 2004), wild radish (*Raphanus raphanistrum* L.) in Australia (Hashem et al. 2001a), paddy weed (*Lindernia micrantha* D.) in Japan (Itoh et al. 1999), and weeds in field crops in Europe (Hartmann et al. 2000).

Intergroup herbicide resistance can be conferred by a non-target-site mechanism, which commonly is en-

hanced metabolism (De Prado and Franco 2004). Metabolic resistance has been reported much more frequently in grass than broadleaf weeds (Werck-Reichhart et al. 2000). Cases of weed resistance due to metabolic detoxification are more frequent than those attributed to target-site mutation in U.K. populations of blackgrass, *Avena* spp., and Italian ryegrass (*Lolium multiflorum* Lam.) resistant to ACCase inhibitors or other herbicides; in European populations of blackgrass resistant to ACCase inhibitors, ALS inhibitors, or chlortoluron; and in European populations of grass species resistant to ALS inhibitors (Claude et al. 2004; Marshall and Moss 2004; Moss et al. 2003). Metabolism-based resistance to herbicides of different sites of action will clearly limit the effectiveness of herbicide group rotation as a tool to delay the evolution of herbicide resistance. Testing populations to determine herbicide resistance patterns is even more important where intergroup resistance is suspected and will help identify remaining herbicide options for farmers (Beckie et al. 2000).

Herbicides that are not readily metabolized in weeds are less likely to select for metabolism-based resistance. For example, the low incidence of dinitroaniline (e.g., trifluralin) resistance may be due to the paucity of detoxification mechanisms in target plants (Holt et al. 1993). Sulfometuron and imazapyr are slowly metabolized in plants and have been used to discriminate between target-site and metabolic resistance in rigid ryegrass (Boutsalis and Powles 1995; Preston and Powles 2002b). Two major enzyme systems have been implicated in herbicide resistance due to increased detoxification—cytochrome P450 monooxygenases and glutathione *S*-transferases (Table 2). These detoxification systems are expressed both constitutively and induced (upregulated) in response to herbicide safeners. Studies of the inheritance of cytochrome P450 monooxygenase-dependent resistance in weeds have shown that a single gene can endow cross-resistance to herbicides of different sites of action applied at registered rates (Letouzé and Gasquez 2001; Preston 2004). Cross-resistance can frequently occur between ACCase and ALS inhibitors, or between photosystem-II inhibitors and ACCase inhibitors (Preston 2004). However, different patterns of cross-resistance can occur in different species (Preston and Mallory-Smith 2001).

Herbicides used in sequences or rotations that are detoxified via pathways different from these two enzyme systems, or that are slowly or not metabolized (e.g., glyphosate, glufosinate, paraquat), will reduce the risk of selecting for metabolism-based, intergroup-HR weed

Table 2. Herbicides metabolized by cytochrome P450 monooxygenases (P450s) or glutathione S-transferases (GSTs) in herbicide-resistant weed biotypes.

Species	P450s	GSTs	Herbicide	Chemical class ^a	Reference
Rigid ryegrass (<i>Lolium rigidum</i> Gaudin)	X		Simazine	Triazine	Burnet et al. (1993a)
	X		Chlortoluron	Urea	Burnet et al. (1993b)
	X		Chlorsulfuron	SU	Christopher et al. (1994)
	X		Diclofop	APP	Preston et al. (1996)
	X		Pendimethalin	Dinitroaniline	Tardif and Powles (1999)
Italian ryegrass (<i>Lolium multiflorum</i> Lam.)	X		Chlorsulfuron	SU	Bravin et al. (2004)
Blackgrass (<i>Alopecurus myosuroides</i> Huds.)	X		Chlorotoluron	Urea	Kemp et al. (1990)
	X		Isoproturon	Urea	Kemp et al. (1990)
	X		Diclofop	APP	Menendez and De Prado (1996)
		X	Fenoxaprop-P	APP	Cummins et al. (1997)
	X		Fenoxaprop-P	APP	Letouzé and Gasquez (2001)
	X		Flupyr-sulfuron	SU	Letouzé and Gasquez (2003)
	X		Isoproturon	Urea	Letouzé and Gasquez (2003)
	X		Chlorproturon	Urea	Letouzé and Gasquez (2003)
	X		Haloxypfop	APP	Letouzé and Gasquez (2003)
	X		Clodinafop	APP	Letouzé and Gasquez (2003)
	X		Diclofop	APP	Maneechote et al. (1997)
	X		Isoproturon	Urea	Singh et al. (1998)
			X	Atrazine	Triazine
Sterile oat (<i>Avena sterilis</i> L.)					
Littleseed canarygrass (<i>Phalaris minor</i> Retz.)	X		Isoproturon	Urea	Singh et al. (1998)
Foxtail (<i>Setaria</i> spp.)		X	Atrazine	Triazine	De Prado et al. (1999)
Late watergrass [<i>Echinochloa phyllopogon</i> (Stapf) Koss.]	X		Bispyribac	PTB	Fischer et al. (2000)
Downy brome (<i>Bromus tectorum</i> L.)	X		Propoxycarbazone ^b	SCT	Park et al. (2004)
Large crabgrass [<i>Digitaria sanguinalis</i> (L.) Scop.]	X		Imazethapyr	IMI	Hidayat and Preston (2001)
Common chickweed [<i>Stellaria media</i> (L.) Vill.]	X		Mecoprop	Phenoxy	Coupland et al. (1990)
Velvetleaf (<i>Abutilon theophrasti</i> Medicus)		X	Atrazine	Triazine	Anderson and Gronwald (1991)
Wild mustard (<i>Sinapis arvensis</i> L.)	X		Ethametsulfuron	SU	Veldhuis et al. (2000)
Annual sowthistle (<i>Sonchus oleraceus</i> L.)		X	Simazine	Triazine	Fraga and Tasende (2003)

^a Abbreviations: APP, aryloxyphenoxypropionate; IMI, imidazolinone; PTB, pyrimidinylthiobenzoate; SCT, sulfonylamino-carbonyltriiazolinone; SU, sulfonyl-urea.

^b Proposed name. Chemical name: methyl 2({[4-methyl-5-oxo-3-propoxy-4,5-dihydro-1H-1,2,4-triazol-1-yl]carbonyl}amino)sulfonyl)benzoate sodium salt.

biotypes. Most cases of cross-resistance across herbicide sites of action have occurred through the use of wheat-selective herbicides (Hidayat and Preston 2001). Thus, herbicides not selective in wheat, such as sethoxydim or clethodim, and nonselective herbicides used in HR crops, such as glyphosate or glufosinate, will be important tools for managing metabolic resistance in grass weed biotypes in the future.

Herbicide-Resistant Crops: A Double-Edged Sword.

Adoption of HR crops is driven primarily by easier and improved weed control or higher net returns (Burnside 1992; Devine and Buth 2001). Cultivation of such crops will increasingly influence future herbicide-use patterns. Globally, resistance to nonselective herbicides (i.e., glyphosate, glufosinate) is the dominant type of transgenic crop (72%, stacked traits excluded), and cultivated area has continued to expand since 1995 (Table 3). HR soybean [*Glycine max* (L.) Merr.] comprises the largest area at 48.4 million ha or 60% of the area planted to transgenic crops. Other important transgenic-HR crops include corn (*Zea mays* L.) (10% by area), cotton (about 6%), and canola (6%). Worldwide, 56% of soybean, 19%

of canola, 15% of cotton, and 6% of corn planted in 2004 were transgenic-HR cultivars.

The judicious use of HR crops can slow the selection of HR weeds by increasing herbicide rotation options, such as the substitution of high-risk herbicides with lower-risk products. Nonselective herbicides used in HR crops in North America have been a powerful tool to proactively and reactively manage HR weeds, such as those resistant to high-risk herbicides, including ACCase and ALS inhibitors (Beckie et al. 2006). As a result, the potential economic impact of these HR weeds has been diminished. However, frequent use of HR crops in cropping systems, resulting in recurrent application of herbicides of the same site of action, may select for new HR weed biotypes or augment the selection that has occurred previously. Evolved weed resistance through selection pressure in HR crops generally poses a greater risk than evolved resistance in related weed species through gene flow because frequency of interspecific hybridization and subsequent introgression is often low (Beckie et al. 2001c; Warwick et al. 1999, 2004). Notable exceptions may include gene flow from HR canola to bird's rape/field mustard (*Brassica rapa* L.) in eastern

Table 3. Transgenic^a crops grown in 2003 and 2004, listed by country, trait, and crop (adapted from James 2003, 2004).

	2003		2004	
	ha × 10 ⁶	%	ha × 10 ⁶	%
By country:				
United States	42.8	63	47.6	59
Argentina	13.9	21	16.2	20
Canada	4.4	6	5.4	6
Brazil	3.0	4	5.0	6
China	2.8	4	3.7	5
Paraguay	0	0	1.2	2
India	0.1	<1	0.5	1
South Africa	0.4	1	0.5	1
Other	0.3	<1	0.4	<1
Total countries	18		17	
Total area	67.7		81.0	
By trait:				
Herbicide resistance (HR)	49.7	73	58.6	72
Bt (<i>Bacillus thuringiensis</i>)	12.2	18	15.6	19
HR + Bt	5.8	9	6.8	9
By crop:				
Soybean [<i>Glycine max</i> (L.) Merr.] ^b	41.4	61	48.4	60
Corn (<i>Zea mays</i> L.) ^c	15.5	23	19.3	23
Cotton (<i>Gossypium hirsutum</i> L.) ^d	7.2	11	9.0	11
Canola (<i>Brassica napus</i> L.) ^e	3.6	5	4.3	6

^a Imidazolinone-HR crops excluded.

^b All HR.

^c HR = 6.4 million ha (9%) in 2003 and 8.1 million ha (10%) in 2004.

^d HR = 4.1 million ha (6%) in 2003 (data not available for 2004).

^e All HR.

Canada (Warwick et al. 2003), HR wheat to jointed goat-grass (*Aegilops cylindrica* Host) in the western United States (Hanson et al. 2005; Seefeldt et al. 1998; Zemetra et al. 1998), and HR rice (*Oryza sativa* L.) to red rice (*O. sativa* L.) in the Americas (Gealy et al. 2003).

Potential impact of HR crops on selection for weed resistance is largely dependent on the size and intensity of the cropped area in an agricultural region and the herbicide site of action. Occurrence of glufosinate-HR weeds has not been reported. There are relatively few reports of weeds resistant to photosynthesis inhibitors at photosystem II (benzotrioles) (Heap 2005). The largest class of HR weeds worldwide are those resistant to ALS inhibitors. The use of ALS inhibitor herbicides in imidazolinone-HR crops will continue the selection for ALS inhibitor-HR broadleaf and grass weeds. Unless imidazolinone-HR crops and ALS inhibitor herbicides are used wisely, their commercial success will be limited.

Since the introduction of glyphosate-HR crops in the mid-1990s, several weed species resistant to the herbicide have been reported (Heap 2005). The majority of glyphosate-HR biotypes were not a consequence of glyphosate selection pressure in HR field crop production systems, but in orchards and vineyards, roadsides, or non-HR crops (e.g., preplant, preharvest, or postharvest).

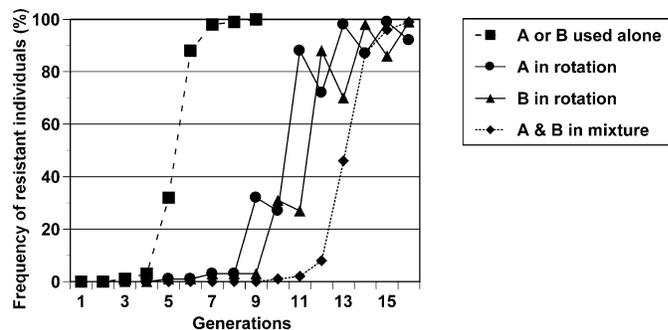


Figure 2. Predicted evolution of herbicide resistance (dominant inheritance) in an outcrossing weed species following repeated selection with herbicides A and B used alone, in a rotation, or in a mixture (adapted from Powles et al. 1997).

To date since 2000, however, evolution of three glyphosate-HR biotypes has been linked to glyphosate-HR cropping systems in the United States. In various regions of the United States, sequential in-season applications combined with near glyphosate-HR soybean monoculture (or glyphosate-HR cotton) have contributed to the evolution of glyphosate-HR horseweed across a large area in more than 10 states, glyphosate-HR common ragweed (*Ambrosia artemisiifolia* L.) in Missouri, and glyphosate-HR Palmer amaranth (*Amaranthus palmeri* S. Wats.) in Georgia (Heap 2005). Such practices create an intense selection pressure for weed resistance and jeopardize the future utility of this important herbicide. Given the importance of glyphosate in reduced-tillage cropping, monoculture glyphosate-HR crops and multiple in-crop glyphosate applications should be dissuaded. The inexpensive cost of glyphosate relative to total variable costs and its lack of soil residual activity are disincentives for a reduction in herbicide-use intensity. Nevertheless, greater implementation of IWM practices in glyphosate-HR crops, such as an intermediate (e.g., 38 cm) rather than a wide (e.g., 76 cm) row spacing in soybean (Chandler et al. 2001), can reduce weed populations and thus help reduce the real or perceived need for sequential in-crop glyphosate applications.

Herbicide Mixtures. Based on the compounded resistance frequency model, herbicide mixtures are predicted to delay resistance longer than rotations (Diggle et al. 2003; Powles et al. 1997) (Figure 2). Field experiments are being conducted to verify model predictions (H. Beckie, unpublished data). Acceptance by farmers of herbicide mixtures for resistance avoidance has been aided by cost-incentive programs from industry, formulated mixtures (e.g., phenoxy plus an ALS inhibitor), and the rapid evolution of resistance in specific cases. The herbicide combinations may be applied at lower individual

herbicide rates (Little and Tardif 2005), especially when interacting synergistically (Gressel 1990). A survey of 1,800 farmers in western Canada from 2001 to 2003 indicated that a majority of them tank-mix herbicides to delay or manage ALS inhibitor-HR broadleaf weeds (H. Beckie, unpublished data).

If mixing partners of different sites of action do not meet the criteria of similar efficacy and persistence, plus different propensity for selecting for resistance in target species, the effectiveness of mixtures for delaying target-site resistance will be reduced. For example, a mixture of an ALS inhibitor, chlorimuron, and metribuzin for ALS inhibitor resistance management of common waterhemp in the mid-western United States is not effective because chlorimuron is more persistent than metribuzin and common waterhemp has uneven and season-long emergence (Sprague et al. 1997). Imazaquin applied with pendimethalin did not delay imazaquin resistance in smooth pigweed because pendimethalin did not adequately control the species (Manley et al. 1998). Mixtures can inadvertently accelerate the evolution of multiple resistance if they fail to meet basic criteria for resistance management and are applied repeatedly (Rubin 1991). A biotype of rigid ryegrass became resistant to a mixture of amitrole and atrazine after 10 yr of widespread and repeated use (Burnet et al. 1991). To effectively delay metabolic resistance, the mixing partners must be degraded via different biochemical pathways (Wrubel and Gressel 1994). However, information on the mode of degradation of herbicides in plants is not known by farmers. Furthermore, mixtures to prevent or delay metabolic resistance in grass weeds, where this mechanism is most prevalent, may be cost-prohibitive unless graminicide partners interact synergistically and can be applied at lower rates.

Challenges to farmer adoption of mixtures for herbicide resistance management include increased cost and availability of suitable mixing partners that meet the criteria outlined above. The inherent limitation of mixtures in delaying target-site resistance is illustrated by the following example. The ALS inhibitor herbicide, thifensulfuron plus tribenuron (formulated mixture), is popular for controlling broadleaf weeds in cereal crops in the northern Great Plains. The phenoxy herbicide, MCPA, is registered as a tank mixture with this ALS inhibitor (Anonymous 2005). Eleven weed species are controlled by both mixing partners, including ball mustard [*Neslia paniculata* (L.) Desv.], kochia, redroot pigweed, Russian thistle (*Salsola iberica* Sennen & Pau), field pennycress (*Thlaspi arvense* L.), and wild mustard (*Sinapis arvensis*

L.). This mixture should markedly delay ALS inhibitor target-site resistance in these species, particularly those that are highly self-pollinated, such as field pennycress. MCPA poses a low risk for selecting for resistance (Figure 1), both mixing partners have short soil residual activity, and MCPA is inexpensive. However, the rate of MCPA used in the mixture may result in reduced efficacy on some species, such as redroot pigweed and Russian thistle, compared with that of the ALS inhibitor herbicide. Moreover, common chickweed [*Stellaria media* (L.) Vill.] and common hempnettle (*Galeopsis tetrahit* L.) are only controlled by the sulfonylurea herbicide. Numerous ALS inhibitor-HR populations of these two species have been reported.

There is limited anecdotal evidence of the usefulness of mixtures in herbicide-resistance management. Mixtures with ALS inhibitors have successfully delayed ALS inhibitor resistance in weeds in rice in Japan and in field crops in Europe (Gressel 1997; Itoh et al. 1999). Farmers who included mixtures of herbicides with different sites of action coupled with various cultural practices were less likely to select ALS inhibitor-HR weed populations (Shaner et al. 1997). *Chenopodium* and *Amaranthus* spp., which often have evolved triazine resistance when triazines were used alone, rarely have been reported to evolve resistance where atrazine plus chloracetamide mixtures were used for over 20 yr in monoculture corn in North America (Wrubel and Gressel 1994). Atrazine is applied at a lower rate in this mixture, thus reducing selection pressure. Pendimethalin is an effective mixing partner (or when used in sequence) for propanil to delay or manage propanil resistance in junglerice [*Echinochloa colona* (L.) Link] in rice in Central America (Riches et al. 1997; Valverde 1996).

Effective resistance management is realized by herbicide mixtures that result in synergistic effects. Some carbamates and organophosphates competitively inhibit aryl acylamidase (EC 3.1.1.a), the enzyme responsible for catalyzing propanil metabolism in rice and propanil-HR junglerice. This inhibition can result in synergistic effects. A formulation of propanil and piperophos, a phosphoric herbicide, was first marketed in 1995 in Costa Rica and cost-effectively controls propanil-HR junglerice while achieving selectivity in rice (Valverde 1996; Valverde et al. 1999). Mixtures comprising a reduced rate of propanil and piperophos or anilofos are now widely used in Costa Rica and Columbia (Valverde et al. 2000). Similarly, mixtures of anilofos or piperophos with propanil at various rate combinations synergistically control propanil-HR barnyardgrass in rice in

Table 4. Propanil in combination with piperophos for selective control of propanil-resistant barnyardgrass in rice: additive (A) or synergistic (S) effects (adapted from Norsworthy et al. 1999a).

Propanil	Piperophos	Weed control	Rice injury
		%	
kg ai/ha			
0.83	0	33	0
	0.11	43 A	0
	0.33	42 A	0
	1.0	56 A	0
	3.0	63 S	1
1.65	0	53	0
	0.11	64 A	0
	0.33	57 A	1
	1.0	81 S	4
	3.0	86 S	3
3.3	0	62	0
	0.11	78 A	1
	0.33	83 A	3
	1.0	96 S	3
	3.0	93 S	4
6.6	0	81	0
	0.11	92 A	3
	0.33	94 A	5
	1.0	99 A	8
	3.0	98 A	7
LSD (0.05)		14	3

the southern United States with little or no crop injury (Daou and Talbert 1999; Norsworthy et al. 1999a, 1999b; Talbert et al. 2000) (Table 4).

Can herbicide rotations or mixtures exploit reduced fitness of herbicide-resistant weeds and negative cross-resistance? Reduced fitness of triazine-HR plants compared with HS plants, documented frequently in the 1970s, resulted in optimistic predictions that this “cost of resistance” would also be prevalent in biotypes resistant to herbicides of other sites of action (Gressel and Segel 1982). The target-site mutations conferring most cases of triazine resistance reduce photosynthetic efficiency, which is often manifested by decreased plant productivity and competitiveness (i.e., reduced fitness). Upon discontinuation of triazine herbicides, reduced fitness of HR compared with HS biotypes was predicted to reverse the evolution of resistance at a rate dependent on the fitness differential between biotypes. Unfortunately, reduced fitness of biotypes resistant to herbicides of other sites of action has generally been minimal or not detectable (Holt and Thill 1994). Lack of measurably reduced fitness in HR biotypes has been inferred from little decline in the proportion of HR:HS individuals measured in fields over time after use of the selecting herbicide was discontinued (Andrews and Morrison 1997). For noncompetitive inhibitors of target-site enzymes, such as ACCase or ALS, the various sites of mutations for resistance are not near the active site of the enzyme and thus there is little fitness loss detectable

due to lower affinity for the normal substrates (Gressel 1999; Wrubel and Gressel 1994).

Negative cross resistance, that is, HR plants are more sensitive to a herbicide than HS plants, has been documented in several triazine-HR weed biotypes (Dabaan and Garbutt 1997; Gadamski et al. 2000; Jordon et al. 1999; Parks et al. 1996). Some herbicides that inhibit photosystem II bind more efficiently to the mutant triazine binding domain than to the wild (HS) type. Triazine-HR weeds frequently show negative cross resistance to other photosystem-II inhibitors, such as bentazon and pyridate; triazine-HR weeds can also exhibit negative cross resistance to herbicides that do not affect photosystem II (Gadamski et al. 2000). Explanations for this phenomenon depend on the specific herbicide, but are largely speculative. The potential combined value of negative cross-resistance and general lack of fitness of triazine-HR biotypes in managing triazine resistance in weeds worldwide has yet to be realized (Gadamski et al. 2000). Nevertheless, pyridate is now mixed with triazine herbicides and applied on millions of hectares annually, especially in Europe, to control triazine-HR biotypes and preserve the cost-effectiveness of this class of herbicides (Gressel 2002). Negative cross resistance has also been observed in non-triazine-HR biotypes. For example, an imidazolinone-HR smooth pigweed biotype was 10-fold more sensitive to cloransulam, another ALS inhibitor, compared with an HS biotype (Poston et al. 2001).

Herbicide Rates. Many herbicides are commonly applied at less-than-registered rates to reduce costs. For example, in-crop herbicides are applied at reduced rates to 28% of cropped land annually in western Canada (Leeson et al. 2004, 2006; Thomas et al. 2003). When farmers apply herbicides at below-registered rates, it is based primarily on their experience with a product’s performance as affected by weed growth stage or environmental conditions. They expect good weed control, although they are aware of the increased risk of suboptimal control. However, herbicide rate reduction without a corresponding reduction in efficacy will have no effect on selection for resistance. Model simulations have suggested that it is not profitable to reduce herbicide rates to reduce selection pressure (efficacy or persistence) for resistance, unless accompanied by a compensating increase in nonherbicidal weed control (Diggle and Neve 2001). The resulting increase in the abundance of HS weed populations would reduce crop yield and quality and increase weed seed return to the seed bank (Gordard et al. 1996; Morrison and Friesen 1996).

Beckie and Kirkland (2003) examined the implication

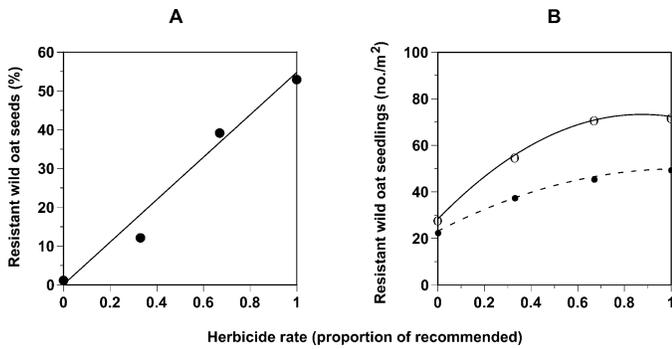


Figure 3. Implication of reduced herbicide rates on target-site resistance enrichment in wild oat: percentage of ACCase inhibitor-resistant individuals in seeds harvested after 4 yr of herbicide application at varying rates (A), and resistant seedlings recruited from the seed bank after 4 yr for the recommended (open circles) and high crop seeding rate (solid circles) treatments (B) [reproduced from Beckie and Kirkland (2003) by permission of the Weed Science Society of America].

of reduced rates of ACCase inhibitors in a 4-yr diverse crop rotation in conjunction with variable crop seeding rates on the enrichment of HR (target-site based) wild oat. As simulation models predict, reduced herbicide efficacy decreased the proportion of HR individuals in the population after 4 yr (Figure 3A). The high crop seeding rate compensated for a one-third reduction in herbicide rate by limiting total (HR plus HS) wild oat seed production and by reducing the number of HR seedlings recruited from the seed bank (Figure 3B). The study concluded that the level of resistance in the seed bank can be reduced without increasing the total seed bank population by manipulating agronomic practices to increase crop competitiveness against wild oat when ACCase inhibitor rates are reduced.

Herbicides applied at registered rates can clearly select for major gene (e.g., target-site) resistance, whereas initially, suboptimal herbicide rates may select for both major and minor gene (i.e. quantitative) resistance. Evolution of quantitative resistance relies on outcrossing among plants, resulting in incremental accumulation in their progeny of minor genes with additive or multiplicative effects (Jaseniuk et al. 1996). Therefore, such herbicide resistance is most probable and would evolve most rapidly in species such as blackgrass, rigid ryegrass, and kochia. Quantitative resistance has been documented or postulated in HR weed populations such as chlortoluron-HR blackgrass in the United Kingdom (Cavan et al. 1999; Chauvel and Gasquez 1994; Hall et al. 1994; Willis et al. 1997), diclofop-HR rigid ryegrass in Australia (Gressel 1997; Neve and Powles 2005a, 2005b; Preston and Powles 2002b), dicamba-HR kochia in North America (Belles et al. 2005; Cranston et al. 2001; Dyer et al. 2000; Westra et al. 2000), and isoproturon-

Table 5. Incremental increase in the level of resistance (resistance factor, R/S), as measured by the dose required to kill 50% of the population (LD_{50}) or reduce biomass by 50% (GR_{50}), of rigid ryegrass biotype VLRI after two or three cycles of selection with diclofop applied at sublethal doses (0.1 to 2 times the $1\times$ rate of 375 g ai/ha) under greenhouse conditions (adapted from Neve and Powles 2005a).

Diclofop selection regime (proportion of $1\times$ rate)	R/S based on LD_{50}	R/S based on GR_{50}
Nontreated control		
0.1, 0.2	7.4	6.7
0.1, 0.2, 0.5	11.8	16.3
0.1, 0.2, 1	55.8	49.3
0.1, 0.5	10.9	6.4
0.1, 0.5, 2	40.1	20.4

HR littleseed canarygrass in India (Kulshrestha et al. 1999; Malik and Singh 1995; Singh et al. 1998, 1999). Less-than-recommended rates have been implicated or speculated as the causal factor in herbicide resistance in these biotypes. These species have a significantly or highly outcrossing mating system, except littleseed canarygrass (Malik et al. 1998).

A population of rigid ryegrass evolved resistance to diclofop at the field-recommended rate when it was exposed to two or three cycles of sublethal rates in the greenhouse (Table 5). Similar results were found in a greenhouse study of the effect of sublethal rates of diclofop on 31 previously nontreated populations of rigid ryegrass (Neve and Powles 2005b). These results were consistent with those of a previous epidemiological study where levels of diclofop resistance in rigid ryegrass populations were positively correlated with the total amount of the herbicide applied over time and where low rates relative to those applied in other countries were typically used (Gressel 1997; Heap and Knight 1982). Maxwell and Mortimer (1994) and Gressel (1997) suggest that soil-residual herbicides may select for quantitative resistance because late-emerging weeds are exposed to lower herbicide doses that may allow accumulation of HR alleles. However, the mechanism of resistance to soil residual herbicides, such as triazines and sulfonyleureas, is often target-site (i.e., major gene) mutation.

Gressel (1995) and Gardner et al. (1998) advocated a tactic of revolving herbicide doses to delay the evolution of major monogene (target site) and quantitative resistance. Routine reduced-rate application that lowers efficacy is not a good weed- or weed-resistance-management tactic (Morrison and Friesen 1996). If suboptimal rates are applied, nonherbicide methods to suppress weed seed production should be employed. Clearly, herbicides should not be repeatedly applied at suboptimal

rates to significantly or highly outcrossing target weeds, such as *Lolium* spp., blackgrass, and kochia, particularly when they occur in large populations (Gressel 2002; Jasieniuk et al. 1996).

Are there opportunities, however, to reduce rates without significantly lowering herbicide efficacy? In the past, registered rates were frequently based on the amount needed to control the least-sensitive weed, whereas other weeds on the product registration may be sensitive at much lower rates. Thus, the selection pressure on these very sensitive species can be extremely high. For example, reduced but effective ALS inhibitor herbicide rates used to control common chickweed in Europe compared with those used in North America doubled the time for resistance evolution by reducing the time those herbicides remain active in the soil (Beckie et al. 2001a; Kudsk et al. 1995). Recent trends in herbicide regulation and registration include more detailed information provided to users to adjust rates according to prevailing environment conditions and herbicide sensitivity, growth stage, or population densities of the target species; a primary regulatory objective in many countries is to promote the application of products at minimum effective doses (N. Malik, personal communication).

Site-Specific Herbicide Application. Site-specific management with the use of a global positioning system can be useful in monitoring and managing HR weed patches at early stages of development in a field over time. Unfortunately, most farmers in the northern Great Plains fail to detect small HR patches (H. Beckie, unpublished data). Comprehensive field scouting and HR weed patch management after in-crop herbicide application are usually not performed because of either a lack of awareness of the benefit of this practice or inconvenience due to large farm size. A study conducted at a 64-ha no-till site in western Canada assessed how preventing seed shed from HR wild oat affected patch expansion over a 6-yr period (Beckie et al. 2005). Area of treated patches increased by 35%, whereas nontreated patches increased by 330% (Figure 4). Patch expansion was attributed mainly to natural seed dispersal (nontreated) or seed movement by equipment at time of planting (nontreated and treated). Extensive (94 to 99%) seed shed from plants in nontreated patches before harvest or control of HR plants by alternative herbicides minimized seed movement by the combine harvester. Although both treated and nontreated patches were relatively stable over time, this study demonstrated that preventing seed production and shed in HR wild oat patches can markedly slow the rate of patch expansion. Consequently, herbi-

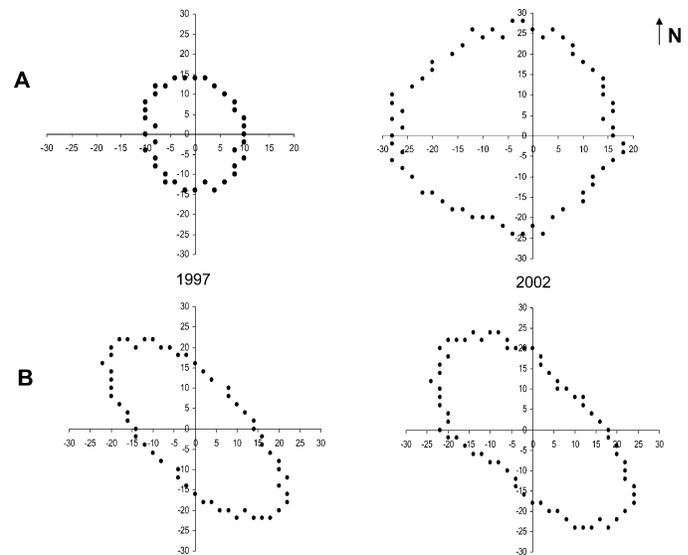


Figure 4. Patch management of herbicide-resistant wild oat in a 6-yr experiment in the northern Great Plains: a nontreated patch in 1997 and 2002 (A) vs. seed shed prevented in a patch from 1997 to 2002 (B) (x and y axis in meters, adapted from Beckie et al. 2005).

cide effectiveness in a field is extended in space and time.

Site-specific herbicide application, utilizing weed abundance as a basis for delineating application areas in a field, would allow some reduction in the overall selection pressure. Costs of acquiring reliable weed-abundance distribution maps and herbicide application have limited its adoption by dry-land farmers growing relatively low cash-value crops. The effect of precision herbicide application on the rate of evolution of resistance would depend on the frequency of herbicide application to specific areas of a field over time and the proportion of the field treated each year. If application frequency of herbicides to specific areas of a field (e.g., lower-slope areas) is similar to conventional herbicide application, HR gene (seed, pollen) flow from these field areas to those treated less frequently may negate any potential benefits of the technology. Furthermore, if these treated areas contain the majority of the weed population present in the field, then this tactic may still result in a selection pressure similar to that of a blanket application.

Analogous to the refugia tactic in crops possessing the *Bacillus thuringiensis* trait to mitigate insect resistance, HS weed refuges has been proposed as a tactic to delay the evolution of herbicide resistance. However, leaving refugia of HS individuals to dilute the proportion of HR alleles in a population by gene flow will not be effective because the recessive control of resistance in outcrossing weed species is rare (Jasieniuk et al. 1996). Additionally, in cases of triazine resistance conferred by chloroplast

gene mutation, genetic recombination among plants does not occur (Stankiewicz et al. 2001). The only documented case of recessive inheritance of major monogene resistance in an outcrossing species was that of a picloram-HR yellow starthistle (*Centaurea solstitialis* L.) biotype found in the state of Washington (Sabba et al. 2003; Sterling et al. 2002).

INTEGRATING NONHERBICIDE TACTICS WITH HERBICIDES

Minimizing weed seed production is central to both HR and non-HR weed management programs. Cultural or mechanical practices affect weed population densities and seed production, and thus can delay the evolution of herbicide resistance by reducing the number of HR alleles in a population. Where high levels of HR alleles are believed to be present in unselected populations, such as ALS inhibitor resistance in common waterhemp and Palmer amaranth in North America (Peterson 1999) or *Lolium* spp. in Europe and Australia (Dinelli et al. 2000; Matthews and Powles 1992; Maxwell and Mortimer 1994; Preston and Powles 2002a), it is important to maintain low population densities via nonchemical methods or by using herbicides with a relatively low likelihood to select for these HR alleles. This tactic is also useful in fields where the high-risk ACCase and ALS inhibitors have been used frequently for over 20 yr. Many of these fields are likely well advanced along the herbicide-resistance evolution curve.

Cultural or mechanical practices will only halt or reverse the rate of enrichment for herbicide resistance in a weed population by eliminating selection pressure (controlling HS and HR plants equally in the absence of herbicide selection pressure) or controlling HR plants more than HS plants, respectively. Nonherbicide practices may increase the effective kill of HR plants relative to that of HS plants in situations where differences exist in the population dynamics of HR and HS biotypes. Seeds of triallate-HR wild oat are generally less dormant than those of HS populations (O'Donovan et al. 1999). Greater and more rapid emergence of HR individuals compared with HS individuals, analogous to that of ALS inhibitor-HR kochia biotypes (Dyer et al. 1993), may be potentially exploited for selective HR biotype control by tillage or nonselective herbicides before delayed planting. Similarly, triazine-HR black nightshade (*Solanum nigrum* L.) in The Netherlands emerges earlier than HS plants because of germination at lower soil temperatures (Kremer and Lotz 1998). In contrast, early planting of winter wheat in the Pacific northwest region of the Unit-

ed States can potentially reduce the competitive ability of HR Italian ryegrass, which emerges later than HS individuals (Radosevich et al. 1997). Tillage to bury seeds of an HR biotype of rigid ryegrass inhibited seedling recruitment compared with that of an HS biotype (Vila-Aiub et al. 2005).

The issues of economic risk, labor availability, and time management impact the adoption of some cultural or mechanical practices for HR weed management. Moreover, some practices such as stubble burning or intensive tillage are contrary to recommendations to improve soil or air quality or conserve soil, water, and energy, and thus their use is discouraged. Evolution of herbicide resistance in weed populations often has not resulted in less herbicide use or a marked increase in nonchemical control methods, except in some cases such as intergroup resistance in weeds (Powles et al. 1997; Preston and Mallory-Smith 2001) or glyphosate-HR horseweed (Steckel et al. 2005). Used singly, the effectiveness of nonherbicide practices is lower and less consistent than that of many herbicides, and may be highly dependent on environmental conditions; when used in combination, however, nonherbicide practices can manage weeds effectively (Blackshaw et al. 2004; Gill and Holmes 1997; Matthews 1994) (Table 6). Some nonherbicide tactics and practices that have proven effective in managing HR weeds are summarized below.

Cropping Systems and Practices. Crop rotations are dictated primarily by profit potential and not the management of HR weeds. Crop rotation, however, is frequently cited as one of the most influential factors in delaying or managing HR weeds (Bourgeois et al. 1997b; Carey et al. 1995; Chauvel et al. 2001; Gill and Holmes 1997; Hartmann et al. 2000; Powles et al. 1997; Ritter and Menbere 1997; Shaner et al. 1999; Singh et al. 1999; Stephenson et al. 1990). Diversity in sequences of crop types and phenologies in a rotation (i.e., dicots vs. monocots; winter- vs. spring-planted; cool vs. warm season; annual vs. perennial) may directly or indirectly reduce weed populations. Crop rotations can facilitate herbicide rotation or reduction (Beckie and Gill 2006). A field study in the northern Great Plains linked ACCase and ALS inhibitor resistance in wild oat to a lack of crop rotation diversity (Beckie et al. 2004). Inclusion of fall-planted and perennial forage crops in annual spring crop-based rotations effectively slowed the evolution of herbicide resistance in this weed species (Figure 5). A field survey documented the ability of 3- to 6-yr alfalfa (*Medicago sativa* L.) stands to reduce wild oat populations in cropping systems through crop competition and cutting

Table 6. Effect of cropping system on density of ACCase inhibitor-resistant blackgrass in the final year of an experiment in Burgundy, France (adapted from Chauvel et al. 2001).

Crop rotation ^a	Tillage ^b	Planting date ^c	Herbicide use ^d	Density
WB-WW-WW	Chisel all	Early	High	no./m ² 9.3
	Moldboard all	Delayed	High	0.8
SB-SP-WW	Chisel all	Delayed	Low	29
	Chisel all	Early	High	1.9
	Chisel-chisel-moldboard	Delayed	High	0.3–1.4 ^e
	Chisel all	Delayed	Low	10

^a Winter barley–winter wheat–winter wheat (WB–WW–WW) vs. spring barley–spring pea–winter wheat (SB–SP–WW).

^b Tillage regime (plowing) after crop harvest.

^c Relative to the local area.

^d Relative intensity of use of alternative herbicides.

^e Split treatments consisting of postemergence nitrogen fertilization at low and normal rates, respectively.

regime of the crop for hay (Ominski et al. 1999). The survey found that wild oat population densities were reduced by 96% in cereal fields that followed alfalfa versus a cereal crop.

Traditionally, Australian agriculture was based on crop–pasture rotation systems. A 3-yr pasture phase was shown to be a low-economic-risk option (Gill and Holmes 1997; Pearce and Holmes 1976). Rigid ryegrass population density was reduced 88 to 96% in wheat following pasture grazed in the spring during the flowering and reproduction stages of the weed (Pearce and Holmes

1976). The combination of grazing and nonselective herbicides (spray topping) reduces rigid ryegrass seed production, resulting in a rapid and marked decline in weed abundance (Gill and Holmes 1997). Preference for continuous annual cropping systems and poor economic returns, however, have led to a decline in the widespread inclusion of pastures in rotations (Monjardino et al. 2004).

The potential value of crop rotation to delay or manage HR weeds will not be realized unless accompanied by diversification or reduction in herbicide use. Repeated use of herbicides with the same site of action will negate the weed-suppression benefits associated with crop rotation. Crop rotations had little influence on occurrence of ACCase inhibitor–HR wild oat in the northern Great Plains because farmers frequently applied these herbicides to cereal, oilseed, and annual legume crops that dominate cropping systems (Légère et al. 2000). Occurrence of resistance in wild oat was the lowest in rotations where frequency of fallow was the highest because of the reduced frequency of herbicide use. Similarly, despite diversity in crop rotations in Western Australia, repeated triazine use in different crops selected for triazine resistance in wild radish (Hashem et al. 2001b).

Inclusion of competitive crops and competitive cultivars of a crop in rotations is viewed by farmers as being important in HR weed management (Bourgeois et al. 1997b; Shaner et al. 1999). Quantitative trait loci for traits in wheat associated with weed competitiveness have been identified. These markers can be used by crop breeders to select for weed-competitive genotypes (Coleman et al. 2001). However, crop competitiveness can also be enhanced by increasing seeding rates. With the widespread appearance of HR rigid ryegrass, many Australian farmers are routinely increasing crop seeding rates by 20 to 40%, resulting in greater plant densities,

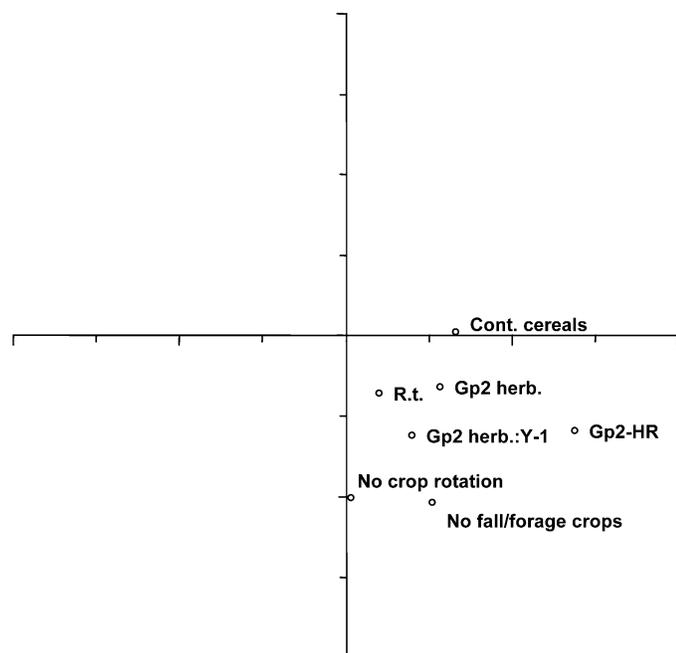


Figure 5. Significant associations between ALS inhibitor-resistant wild oat (Gp2-HR) and management practices in the northern Great Plains as determined from multiple correspondence analysis (Cont. cereals, continuous cereals; R.t., reduced tillage; Gp2 herb., ALS inhibitor used in current year; Gp2 herb.:Y-1, ALS inhibitor used 1 yr before; No crop rotation, crop rotation not used; No fall/forage crops, fall-planted or forage crops not used) (adapted from Beckie et al. 2004).

to improve competitiveness (Table 1) (Gill and Holmes 1997; Medd et al. 1987; Powles 1997). This practice is most cost effective for cereals. In the northern Great Plains, increased crop seeding rate is the most consistent cultural practice for managing weeds and maintaining crop yields (Beckie and Kirkland 2003; Blackshaw et al. 2004).

Delayed planting is often promoted for the control of some HR grass weed species in Europe, such as winter wild oat (*Avena ludoviciana* Durieu), hood canarygrass (*Phalaris paradoxa* L.), and blackgrass (Table 6, Chauvel et al. 2001; Orson 1999; Sattin et al. 2001), by depleting the seed bank before crop planting. Delayed rice planting in Central America is commonly used to reduce HR junglerice population densities (Valverde et al. 2000, 2001). In Australia, delayed crop planting has been integrated with other control tactics to manage HR rigid ryegrass (Table 1) (Gill and Holmes 1997; Powles and Matthews 1996).

Tillage Systems. Owen (2001b) reviewed the impact of tillage and mechanical practices in managing HR weed populations. The judicious use of timely tillage has been cited often as an important practice to delay or manage HR weeds (Bourgeois et al. 1997b; Chauvel et al. 2001; Orson and Livingston 1987; Peterson 1999; Stephenson et al. 1990). Tillage may substitute for herbicide use or influence seed bank dynamics. For example, plowing to bury weed seeds of blackgrass to reduce germination and emergence has been proven highly effective for management of HR populations in Europe (Moss 1997; Orson and Livingston 1987). Timely tillage can also stimulate weed germination before crop planting, such as “autumn tickle” (Table 1) (Boutsalis and Powles 1998; Gill and Holmes 1997).

Anecdotal field observations have frequently linked herbicide resistance in weeds to conservation-tillage systems, particularly no-till, which are increasingly being adopted by farmers because of cost and time efficiencies. Ratification of the Kyoto Protocol on greenhouse gas emissions will further encourage farmers to adopt reduced-tillage systems through economic incentives to increase carbon sequestration in soil. In a field study by Beckie et al. (2004), ALS inhibitor–HR wild oat was associated with such systems (Figure 5). Reduced tillage substitutes herbicide use for tillage to varying degrees. Reduced-tillage cropping can increase the abundance of specific weed species and consequently, result in greater herbicide use. However, an analysis of multiple studies found little evidence that reduced tillage increases herbicide use (Nazarko et al. 2005; Zhang et al. 2000). In

the absence of tillage, weed seedlings may be derived largely from seeds shed in the previous crop and concentrated near the soil surface. Consequently, there will be little buffering against resistance evolution from old seeds, which may have greater percentage susceptibility (Moss 2002).

Limiting Herbicide-Resistance Gene Spread. Gene flow through pollen or seed movement from HR weed populations can provide a source of HR alleles in previously HS populations. Because rates of gene flow are generally higher than rates of mutation, the time required to reach a high level of herbicide resistance in such situations is greatly reduced (Jaseniuk et al. 1996). It is difficult to control the spread of herbicide resistance via pollen flow, especially when resistance is often endowed by a single, dominant or semidominant gene (Letouzé and Gasquez 1999; Richter and Powles 1993; Smeda et al. 2000). For example, pollen of ALS inhibitor–HR kochia can move more than 30 m in a cropped field (Mallory-Smith et al. 1993), and ACCase inhibitor–HR alleles in rigid ryegrass pollen can move more than 10 m in cropped or noncropped conditions (Hawthorn-Jackson et al. 2003). Seed movement is probably responsible for the majority of gene flow in weed populations (Diggle and Neve 2001). Seed movement has the potential to influence HR gene spread on a much larger scale than pollen flow.

HR weed seed spread within and among fields has been documented (Andrews et al. 1998; Hidayat et al. 2004; Li et al. 2000; Ritter and Menbere 1997; Stephenson et al. 1990; Tsuji et al. 2003). Fields within farms are more likely to have HR weeds than randomly picked fields, indicating movement of HR seed between fields via equipment (Anderson et al. 1996) or similar selection pressure among fields within a farm. Sharing of equipment among farmers has also been implicated in herbicide resistance (Debreuil et al. 1996). Weed seed spread by machinery, noncomposted manure, silage, or contaminated commercial seed stocks or feed (Ritter and Menbere 1997; Stephenson et al. 1990) is generally greater than natural seed dispersal. For example, wild oat seeds can spread more than 150 m by a combine harvester (Shirtliffe and Entz 2005). Spread of herbicide resistance among wild oat (*Avena* spp.) patches within 350 m of each other has been documented in the United Kingdom (Cavan et al. 1998). Wind dispersal of weed species having lightweight seeds, such as prickly lettuce (*Lactuca serriola* L.) (Rieger et al. 2001) and horseweed (Dauer and Mortensen 2005; VanGessel 2001), can also spread herbicide resistance rapidly. Wind can efficiently trans-

Table 7. RIM (resistance and integrated management) model scenarios: Economically optimal frequency of integrated weed management (IWM) practices when selective herbicide use is restricted over a 10-year period,^a and resulting plant density of mature rigid ryegrass in a lupin-wheat rotation (adapted from Pannell et al. 2004).

Selective herbicide applications	Optimal frequency of IWM practices ^b				Total usage of nonselective treatments	Mature weed density ^d
	High crop seeding rates	Crop topping ^c	Seed catching	Delayed planting + glyphosate		
No. of years over a 10-yr period					no.	no./m ²
2	10	5	10	10	35	3
4	10	5	10	6	31	6
6	10	4	10	2	26	8
8	10	2	10	1	23	6
10	6	1	10	0	17	6

^a Usage of ACCase- or ALS-inhibiting herbicides restricted for proactive or reactive resistance management.

^b Frequency of use of IWM practices resulting in greatest profitability over a 10-yr period for a given frequency of selective herbicide use.

^c Lupin phase only.

^d 10-yr mean.

port kochia and Russian thistle tumbleweeds for long distances (Mallory-Smith et al. 1993). As the incidence of herbicide resistance increases in a region, pollen and seed movement in addition to selection will increasingly influence such occurrences.

Management practices that limit the spread of HR seed can slow the occurrence of herbicide resistance. In western Canada, farmers who reported practicing weed sanitation (e.g., cleaning harvesting and tillage equipment when moving between fields, covering the grain truck box, mowing or spraying ditches or uncontrolled weed patches, applying composted versus fresh manure) were less likely to have HR wild oat than those who were less careful (Légère et al. 2000). Cleaning equipment when moving among fields, and mowing weed patches, ditches, and headlands ranked fourth and fifth, respectively, in importance among herbicide-resistance-management practices cited by farmers in western Canada (Bourgeois et al. 1997b). If the HR population covers a wide area across the field, management should focus on reducing seed return and spread by using low-risk herbicides in conjunction with cultural practices, such as cutting the crop (hay, silage, or green manure) before or soon after flowering of the HR weed species, growing competitive annual crops such as barley (*Hordeum vulgare* L.) or perennial crops, or collecting weed seeds at harvest. Weed populations can decline rapidly within one to two growing seasons for species having a relatively short-lived seed bank, such as rigid ryegrass.

Capture of weed seeds during the harvest operation is a technique used primarily by farmers dealing with herbicide resistance (Table 1). Some weed species, such as rigid ryegrass, do not shed seeds until well after maturity and therefore allow farmers the opportunity to collect seeds during harvest. In Western Australia, Gill (1996)

reported a 60 to 80% removal of rigid ryegrass seeds, which reduced weed infestation in the subsequent crop by 73%. Although weed seed catching/removal at harvest is effective in managing HR weeds (Gill 1997; Gill and Holmes 1997; Matthews 1994), farmer adoption is low (Powles 1997; Shaner et al. 1999; Thill et al. 1994) (Table 1). In contrast, weeds such as wild oat may shed most seeds by cereal crop harvest in the northern Great Plains. Therefore, harvesting after extensive seed shed can reduce HR wild oat seed spread by equipment (Beckie et al. 2005).

Decision-Support Systems. Use of a decision-support system (DSS) can help farmers choose the best combination of IWM practices to delay or manage HR weeds on their farm. The most advanced DSS to date is the RIM (resistance and integrated management) model developed for IWM of single or multiple species in Australia (Monjardino et al. 2003; Pannell et al. 2004). It allows farmers to quickly assess the agronomic and economic performance of numerous combinations of management options over varying time frames (Table 7). Such a DSS, when continually maintained and updated, can be a useful tool for farmers to combat herbicide resistance in weeds.

CONCLUSIONS

Proactive or reactive management for herbicide resistance in weeds (a) must consider the relative risks of herbicides of different sites of action to select for target-site resistance and the differing propensity of herbicides to be metabolized in HR biotypes when sequencing or rotating herbicides; (b) must meet basic criteria for effective herbicide mixtures; and (c) should incorporate

agronomic practices in cropping systems that help reduce weed seed production and spread. Use of low-risk, non-selective herbicides applied preplant or in HR crops has improved HR weed management. However, frequent use of HR crops such as those resistant to imidazolinones or glyphosate may maintain conditions that lead to resistance, namely, simplified cropping systems favoring a few dominant weed species and frequent use of single site-of-action herbicides.

The extent to which farmers alter their current farming systems to manage herbicide resistance depends on the nature and magnitude of infestation of an HR biotype. In many cases, simply switching to an alternative herbicide will cost-effectively control the HR population. For serious herbicide resistance problems, for example, heavy infestations of intergroup-HR weed species, a longer-term cropping systems approach may be required. Approaches to IWM differ, depending on agroecological conditions, biology, and ecology of the weed species with evolved resistance, and agronomic and socio-economic considerations by farmers. Although herbicides remain the dominant weed-control tool, diversification in cropping systems and practices can result in less herbicide used and thus a reduction in selection pressure for resistance. Even serious weed-resistance problems can be managed successfully if farmers are receptive to changes in their cropping systems. The increasing incidence and complexity of herbicide resistance in weeds will inevitably require farming systems with a reduced dependence on herbicides.

LITERATURE CITED

- Anderson, D. D., F. W. Roeth, and A. R. Martin. 1996. Occurrence and control of triazine-resistant common waterhemp (*Amaranthus rudis*) in field corn (*Zea mays*). *Weed Technol.* 10:570–575.
- Anderson, M. P. and J. W. Gronwald. 1991. Atrazine resistance in a velvetleaf (*Abutilon theophrasti*) biotype due to enhanced glutathione S-transferase activity. *Plant Physiol.* 96:104–109.
- Andrews, T. S. and I. N. Morrison. 1997. The persistence of trifluralin resistance in green foxtail (*Setaria viridis*) populations. *Weed Technol.* 11: 369–372.
- Andrews, T. S., I. N. Morrison, and G. A. Penner. 1998. Monitoring the spread of ACCase inhibitor resistance among wild oat (*Avena fatua*) patches using AFLP analysis. *Weed Sci.* 46:196–199.
- Anonymous. 2005. Guide to Crop Protection: Weeds, Plant Diseases, Insects. Bi-provincial publication. Regina, SK: Saskatchewan Agriculture, Food and Rural Revitalization; Winnipeg, MB: Manitoba Agriculture, Food and Rural Initiatives. 354 p.
- Australian National Glyphosate Sustainability Working Group. 2005. Keeping Glyphosate Resistance Rare in Australian Cropping. Web page: http://www.weeds.crc.org.au/documents/glyphosate_risk_guide_colour.pdf. Accessed: 31 May 2005.
- Beckie, H. J., F. Chang, and F. C. Stevenson. 1999a. The effect of labeling herbicides with their site of action: a Canadian perspective. *Weed Technol.* 13:655–661.
- Beckie, H. J. and G. S. Gill. 2006. Strategies for managing herbicide-resistant weeds. In H. P. Singh, D. R. Batish, and R. K. Kohli, eds. *Handbook of Sustainable Weed Management*. Binghamton, NY: The Haworth Press, Inc. In press.
- Beckie, H. J., L. M. Hall, S. Meers, J. J. Laslo, and F. C. Stevenson. 2004. Management practices influencing herbicide resistance in wild oat. *Weed Technol.* 18:853–859.
- Beckie, H. J., L. M. Hall, and B. Schuba. 2005. Patch management of herbicide-resistant wild oat (*Avena fatua*). *Weed Technol.* 19:697–705.
- Beckie, H. J., L. M. Hall, and F. J. Tardif. 2001a. Herbicide resistance in Canada—where are we today? In R. E. Blackshaw and L. M. Hall, eds. *Integrated Weed Management: Explore the Potential*. Sainte-Anne-de-Bellevue, QC: Expert Committee on Weeds. Pp. 1–36.
- Beckie, H. J., L. M. Hall, and F. J. Tardif. 2001b. Impact and management of herbicide-resistant weeds in Canada. Proc. Brighton Crop Protection Conference—Weeds. Farnham, UK: British Crop Protection Council. Pp. 747–754.
- Beckie, H. J., L. M. Hall, and S. I. Warwick. 2001c. Impact of herbicide-resistant crops as weeds in Canada. Proc. Brighton Crop Protection Conference—Weeds. Farnham, UK: British Crop Protection Council. Pp. 135–142.
- Beckie, H. J., K. N. Harker, L. M. Hall, S. I. Warwick, A. Légère, P. H. Sikkema, G. W. Clayton, A. G. Thomas, J. Y. Leeson, G. Séguin-Swartz, and M. J. Simard. 2006. A decade of herbicide-resistant crops in Canada. *Can. J. Plant Sci.* (in press).
- Beckie, H. J., I. M. Heap, R. J. Smeda, and L. M. Hall. 2000. Screening for herbicide resistance in weeds. *Weed Technol.* 14:428–445.
- Beckie, H. J. and F. A. Holm. 2002. Response of wild oat (*Avena fatua*) to residual and non-residual herbicides in canola (*Brassica napus*) in western Canada. *Can. J. Plant Sci.* 82:797–802.
- Beckie, H. J. and S. Jana. 2000. Selecting for triallate resistance in wild oat. *Can. J. Plant Sci.* 80:665–667.
- Beckie, H. J. and K. J. Kirkland. 2003. Implication of reduced herbicide rates on resistance enrichment in wild oat (*Avena fatua*). *Weed Technol.* 17: 138–148.
- Beckie, H. J. and I. N. Morrison. 1993. Effective kill of trifluralin-susceptible and -resistant green foxtail (*Setaria viridis*). *Weed Technol.* 7:15–22.
- Beckie, H. J., A. G. Thomas, and A. Légère. 1999b. Nature, occurrence, and cost of herbicide-resistant green foxtail (*Setaria viridis*) across Saskatchewan ecoregions. *Weed Technol.* 13:626–631.
- Beckie, H. J., A. G. Thomas, A. Légère, D. J. Kelner, R. C. Van Acker, and S. Meers. 1999c. Nature, occurrence, and cost of herbicide-resistant wild oat (*Avena fatua*) in small-grain production areas. *Weed Technol.* 13: 612–625.
- Beckie, H. J., A. G. Thomas, and F. C. Stevenson. 2002. Survey of herbicide-resistant wild oat (*Avena fatua*) in two townships in Saskatchewan. *Can. J. Plant Sci.* 82:463–471.
- Belles, D. S., S. Nissen, S. Ward, and P. Westra. 2005. Genetics and physiology of a dicamba resistance trait in kochia (*Kochia scoparia*). *Weed Sci. Soc. Am. Abstr.* 45:50.
- Blackshaw, R. E., L. J. Molnar, J. R. Moyer, K. N. Harker, G. W. Clayton, and H. J. Beckie. 2004. Integration of cropping practices and herbicides for sustainable weed management. In Proc. Fourth International Weed Science Congress, Durban, S.A. Davis, CA: International Weed Science Society. P. 122.
- Boerboom, C. M. 1999. Nonchemical options for delaying weed resistance to herbicides in Midwest cropping systems. *Weed Technol.* 13:636–642.
- Bourgeois, L., N. C. Kenkel, and I. N. Morrison. 1997a. Characterization of cross-resistance patterns in acetyl-CoA carboxylase inhibitor resistant wild oat (*Avena fatua*). *Weed Sci.* 45:750–755.
- Bourgeois, L., I. N. Morrison, and D. Kelner. 1997b. Field and grower survey of ACCase resistant wild oat in Manitoba. *Can. J. Plant Sci.* 77:709–715.
- Boutsalis, P. and S. B. Powles. 1995. Resistance of dicot weeds to acetolactate synthase (ALS)-inhibiting herbicides in Australia. *Weed Res.* 35:149–155.
- Boutsalis, P. and S. B. Powles. 1998. Seedbank characteristics of herbicide-resistant and susceptible *Sisymbrium orientale*. *Weed Res.* 38:389–395.
- Bradley, K. W. and E. S. Hagood, Jr. 2001. Identification of a Johnsongrass (*Sorghum halepense*) biotype resistant to aryloxyphenoxypropionate and cyclohexanedione herbicides in Virginia. *Weed Technol.* 15:623–627.
- Bravin, F., A. Onofri, G. Zanin, and M. Sattin. 2004. Is malathion a useful tool to infer the chlorsulfuron-resistance mechanism in multi-resistant Italian populations of *Lolium* spp? In Proc. Fourth International Weed

- Science Congress, Durban, S.A. Davis, CA: International Weed Science Society P. 52.
- Burnet, M.W.M., O. B. Hildebrand, J.A.M. Holtum, and S. B. Powles. 1991. Amitrole, triazine, substituted urea, and metribuzin resistance in a biotype of rigid ryegrass (*Lolium rigidum*). *Weed Sci.* 39:317–323.
- Burnet, M.W.M., B. R. Loveys, J.A.M. Holtum, and S. B. Powles. 1993a. Increased detoxification is a mechanism of simazine resistance in *Lolium rigidum*. *Pestic. Biochem. Physiol.* 46:207–218.
- Burnet, M.W.M., B. R. Loveys, J.A.M. Holtum, and S. B. Powles. 1993b. A mechanism of chlorotoluron resistance in *Lolium rigidum*. *Planta* 190: 182–189.
- Burnside, O. C. 1992. Rationale for developing herbicide-resistant crops. *Weed Technol.* 6:621–625.
- Carey, V. F., III, R. E. Hoagland, and R. E. Talbert. 1995. Verification and distribution of propanil-resistant barnyardgrass (*Echinochloa crus-galli*) in Arkansas. *Weed Technol.* 9:366–372.
- Cavan, G., P. Biss, and S. R. Moss. 1998. Herbicide resistance and gene flow in wild-oats (*Avena fatua* and *Avena sterilis* ssp. *ludoviciana*). *Ann. Appl. Biol.* 133:207–217.
- Cavan, G., J. Cussans, and S. R. Moss. 1999. Modelling strategies to prevent resistance in black-grass (*Alopecurus myosuroides*). *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 777–782.
- Chandler, K., A. Shrestha, and C. J. Swanton. 2001. Weed seed return as influenced by the critical weed-free period and row spacing of no-till glyphosate-resistant soybean. *Can. J. Plant Sci.* 81:877–880.
- Chauvel, B. and J. Gasquez. 1994. Relationships between genetic polymorphism and herbicide resistance within *Alopecurus myosuroides* Huds. *Heredity* 72:336–344.
- Chauvel, B., J. P. Guillemin, N. Colbach, and J. Gasquez. 2001. Evaluation of cropping systems for management of herbicide-resistant populations of blackgrass (*Alopecurus myosuroides* Huds.). *Crop Prot.* 20:127–137.
- Christopher, J. T., C. Preston, and S. B. Powles. 1994. Malathion antagonizes metabolism-based chlorsulfuron resistance in *Lolium rigidum*. *Pestic. Biochem. Physiol.* 49:172–182.
- Claude, J. P., A. Didier, P. Favier, and P. P. Thalinger. 2004. Development of a European database for the evolution follow-up of resistant black-grass (*Alopecurus myosuroides* Huds.) populations in cereal crops. *In Proc. Fourth International Weed Science Congress*, Durban, S.A. Davis, CA: International Weed Science Society. P. 48.
- Cocker, K. M., J.O.D. Coleman, A. M. Blair, J. H. Clarke, and S. R. Moss. 2000. Biochemical mechanisms of cross-resistance to aryloxyphenoxypropionate and cyclohexanedione herbicides in populations of *Avena* spp. *Weed Res.* 40:323–334.
- Coleman, R. K., G. S. Gill, and G. J. Rebetzke. 2001. Identification of quantitative trait loci for traits conferring weed competitiveness in wheat (*Triticum aestivum* L.). *Aust. J. Agric. Res.* 52:1235–1246.
- Coupland, D. 1994. Resistance to the auxin analog herbicides. *In S. B. Powles and J.A.M. Holtum*, eds. *Herbicide Resistance in Plants—Biology and Biochemistry*. Boca Raton, FL: CRC Press. Pp. 171–214.
- Coupland, D., P.J.W. Lutman, and C. Heath. 1990. Uptake, translocation and metabolism of mecaprop in a sensitive and resistant biotype of *Stellaria media*. *Pestic. Biochem. Physiol.* 36:61–67.
- 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.
- Cummins, I., S. R. Moss, J. D. Cole, and R. Edwards. 1997. Glutathione transferases in herbicide-resistant and herbicide-susceptible black-grass (*Alopecurus myosuroides*). *Pestic. Sci.* 51:244–250.
- Dabaan, M. E. and K. Garbutt. 1997. Herbicide cross-resistance in atrazine-resistant velvetleaf (*Abutilon theophrasti*) and redroot pigweed (*Amaranthus retroflexus*). *In H. Brown, G. W. Cussans, M. D. Devine, S. O. Duke, C. Fernandez-Quintanilla, A. Helweg, R. E. Labrada, M. Landes, P. Kudsk, and J. C. Streibig*, eds. *Proc. Second International Weed Control Congress*, Copenhagen, Denmark. Flakkebjerg, Slagelse, Denmark: Department of Weed Control and Pesticide Ecology. Pp. 505–510.
- Daou, H. and R. E. Talbert. 1999. Control of propanil-resistant barnyardgrass (*Echinochloa crus-galli*) in rice (*Oryza sativa*) with carbaryl/propanil mixtures. *Weed Technol.* 13:65–70.
- Dauer, J. T. and D. A. Mortensen. 2005. Long-distance wind dispersal of *Conyza canadensis* and management implications. *In P. Bàrberi et al.* (eds.). *Proc. 13th European Weed Research Society Symposium*, Bari, Italy.
- De Prado, R. A. and A. R. Franco. 2004. Cross-resistance and herbicide metabolism in grass weeds in Europe: biochemical and physiological aspects. *Weed Sci.* 52:441–447.
- De Prado, R., N. López-Martínez, and J. Gonzalez-Gutierrez. 1999. Identification of two mechanisms of atrazine resistance in *Setaria faberi* and *Setaria viridis* biotypes. *Pestic. Biochem. Physiol.* 67:114–124.
- 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.* 44:871–878.
- Dellow, J. J., M. Incerti, R. Britton, and A. Bishop. 1997. Herbicide resistance extension strategy for the south eastern wheat belt of New South Wales, Australia. *In H. Brown, G. W. Cussans, M. D. Devine, S. O. Duke, C. Fernandez-Quintanilla, A. Helweg, R. E. Labrada, M. Landes, P. Kudsk, and J. C. Streibig*, eds. *Proc. Second International Weed Control Congress*, Copenhagen, Denmark. Flakkebjerg, Slagelse, Denmark: Department of Weed Control and Pesticide Ecology. Pp. 487–492.
- Délye, C., A. Matějček, and J. Gasquez. 2002. PCR-based detection of resistance to acetyl-CoA carboxylase-inhibiting herbicides in black-grass (*Alopecurus myosuroides* Huds.) and ryegrass (*Lolium rigidum* Gaud.). *Pest Manage. Sci.* 58:474–478.
- Délye, C. and S. Michel. 2005. 'Universal' primers for PCR-sequencing of grass chloroplastic acetyl-CoA carboxylase domains involved in resistance to herbicides. *Weed Res.* 45:323–330.
- Délye, C., C. Straub, A. Matějček, and S. Michel. 2003. Multiple origins for black-grass (*Alopecurus myosuroides* Huds.) target-site-based resistance to herbicides inhibiting acetyl-CoA carboxylase. *Pest Manage. Sci.* 60: 35–41.
- Devine, M. D. and J. L. Buth. 2001. Advantages of genetically modified canola: a Canadian perspective. *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 367–372.
- Diggle, A. J. and P. Neve. 2001. The population dynamics and genetics of herbicide resistance—a modeling approach. *In S. B. Powles and D. L. Shaner*, eds. *Herbicide Resistance and World Grains*. New York: CRC Press. Pp. 61–99.
- 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.
- Dinelli, G., A. Bonetti, and P. Catizone. 2000. Investigation on biodiversity of Italian *Lolium* spp. populations susceptible and resistant to herbicides. *In A. Légère*, ed. *Proc. Third International Weed Science Congress*, Foz do Iguassu, Brazil. Corvallis, OR: International Weed Science Society. P. 146.
- Dyer, W. E., P. W. Chee, and P. K. Fay. 1993. Rapid germination of sulfonylurea-resistant *Kochia scoparia* L. accessions is associated with elevated seed levels of branched chain amino acids. *Weed Sci.* 41:18–22.
- Dyer, W. E., M. A. Jasieniuk, and B. D. Maxwell. 2000. Stress tolerance in *Kochia scoparia* L.: phenotypic plasticity or genetic adaptation? *In A. Légère*, ed. *Proc. Third International Weed Science Congress*, Foz do Iguassu, Brazil. Corvallis, OR: International Weed Science Society. Pp. 147–148.
- Fischer, A. J., D. E. Bayer, M. D. Carriere, C. M. Ateh, and K. O. Yim. 2000. Mechanisms of resistance to bispyribac-sodium in an *Echinochloa phyllopogon* accession. *Pestic. Biochem. Physiol.* 68:156–165.
- Foes, M. J., L. Liu, P. J. Tranel, L. M. Wax, and E. W. Stoller. 1998. A biotype of common waterhemp (*Amaranthus rudis*) resistant to triazine and ALS herbicides. *Weed Sci.* 46:514–520.
- Fraga, M. I. and M. G. Tasende. 2003. Mechanism of resistance to simazine in *Sonchus oleraceus*. *Weed Res.* 43:333–340.
- Friesen, L. F., T. L. Jones, R. C. Van Acker, and I. N. Morrison. 2000. Identification of *Avena fatua* populations resistant to imazamethabenz, flumprop, and fenoxaprop-P. *Weed Sci.* 48:532–540.
- Gadamski, G., D. Ciarka, J. Gressel, and S. W. Gawronski. 2000. Negative cross-resistance in triazine-resistant biotypes of *Echinochloa crus-galli* and *Conyza canadensis*. *Weed Sci.* 48:176–180.
- Gardner, S. N., J. Gressel, and M. Mangel. 1998. A revolving dose strategy to delay the evolution of both quantitative vs major monogene resistances to pesticides and drugs. *Int. J. Pest Manag.* 44:161–180.
- Gealy, D. R., D. H. Mitten, and J. N. Rutger. 2003. Gene flow between red rice (*Oryza sativa*) and herbicide-resistant rice (*O. sativa*): implications for weed management. *Weed Technol.* 17:627–645.
- Gill, G. S. 1995. Development of herbicide resistance in annual ryegrass populations (*Lolium rigidum* Gaud.) in the cropping belt of Western Australia. *Aust. J. Exp. Agric.* 35:67–72.
- Gill, G. S. 1996. Management of herbicide resistant ryegrass in Western Aus-

- tralia—research and its adoption. In R.C.H. Shepherd, ed. Proc. 11th Aust. Weeds Conf. Melbourne, Australia: Weed Science Society of Victoria. Pp. 542–545.
- Gill, G. S. 1997. Prevention and control of herbicide resistant weeds in Australia. In R. De Prado, J. Jorrín, and L. García-Torres, eds. *Weed and Crop Resistance to Herbicides*. London: Kluwer Academic. Pp. 305–313.
- Gill, G. S. and J. E. Holmes. 1997. Efficacy of cultural control methods for combating herbicide-resistant *Lolium rigidum*. *Pestic. Sci.* 51:352–358.
- Gorrdard, R. J., D. J. Pannell, and G. Hertzler. 1996. Economic evaluation of strategies for management of herbicide resistance. *Agric. Syst.* 51: 281–298.
- Gressel, J. 1990. Synergizing herbicides. *Rev. Weed Sci.* 5:49–82.
- Gressel, J. 1995. Creeping resistances: the outcome of using marginally-effective or reduced rates of herbicides. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 587–590.
- Gressel, J. 1997. Burgeoning resistance requires new strategies. In R. De Prado, J. Jorrín, and L. García-Torres, eds. *Weed and Crop Resistance to Herbicides*. London: Kluwer Academic. Pp. 3–14.
- Gressel, J. 1999. Modern herbicide design can lead to obsolescence via resistance. *Weed Sci. Soc. Am. Abstr.* 39:76–77.
- Gressel, J. 2002. *Molecular Biology of Weed Control*. New York: Taylor & Francis. 504 p.
- Gressel, J. and L. A. Segel. 1982. Interrelating factors controlling the rate of appearance of resistance: the outlook for the future. In H. M. LeBaron and J. Gressel, eds. *Herbicide Resistance in Plants*. New York: Wiley. Pp. 325–347.
- Hall, L. M., J.A.M. Holtum, and S. B. Powles. 1994. Mechanisms responsible for cross resistance and multiple resistance. In S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants—Biology and Biochemistry*. Boca Raton, FL: CRC Press. Pp. 243–261.
- Hanson, B. D., C. A. Mallory-Smith, W. J. Price, B. Shafii, D. C. Thill, and R. S. Zemetra. 2005. Interspecific hybridization: potential for movement of herbicide resistance from wheat to jointed goatgrass (*Aegilops cylindrica*). *Weed Technol.* 19:674–682.
- Hartmann, F., I. Lánszki, L. Szentey, and A. Tóth. 2000. Resistant weed biotypes in Hungary. In A. Légère, ed. Proc. Third International Weed Science Congress, Foz do Iguassu, Brazil. Corvallis, OR: International Weed Science Society. P. 138.
- Hashem, A., D. Bowran, T. Piper, and H. Dhammu. 2001a. Resistance of wild radish (*Raphanus raphanistrum*) to acetolactate synthase-inhibiting herbicides in the Western Australia wheat belt. *Weed Technol.* 15:68–74.
- Hashem, A., H. S. Dhammu, S. B. Powles, D. G. Bowran, T. J. Piper, and A. H. Cheam. 2001b. Triazine resistance in a biotype of wild radish (*Raphanus raphanistrum*) in Australia. *Weed Technol.* 15:636–641.
- Hawthorn-Jackson, D., R. Davidson, and C. Preston. 2003. The spread of herbicide resistant annual ryegrass pollen. *Weed Sci. Soc. Am. Abstr.* 43: 76.
- Heap, I. M. 1999. International survey of herbicide-resistant weeds: lessons and limitations. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 769–776.
- Heap, I. M. 2005. International Survey of Herbicide Resistant Weeds. Web page: <http://www.weedscience.org>. Accessed: 2 May 2005.
- Heap, I. and R. Knight. 1982. A population of ryegrass tolerant to the herbicide diclofop-methyl. *J. Aust. Inst. Agric. Sci.* 48:156–157.
- Hidayat, I., J. Baker, and C. Preston. 2004. Evolution and spread of herbicide resistant barley-grass in South Australia. *Weed Sci. Soc. Am. Abstr.* 44: 67.
- Hidayat, I. and C. Preston. 2001. Cross-resistance to imazethapyr in a fluazifop-P-butyl-resistant population of *Digitaria sanguinalis*. *Pestic. Biochem. Physiol.* 71:190–195.
- Holt, J. S., S. B. Powles, and J.A.M. Holtum. 1993. Mechanisms and agronomic aspects of herbicide resistance. *Ann. Rev. Plant Physiol. Plant Mol. Biol.* 44:203–229.
- Holt, J. S. and D. C. Thill. 1994. Growth and productivity of resistant plants. In S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants—Biology and Biochemistry*. Boca Raton, FL: CRC Press. Pp. 299–316.
- Itoh, K., G. X. Wang, and S. Ohba. 1999. Sulfonylurea resistance in *Lindernia micrantha*, an annual paddy weed in Japan. *Weed Res.* 39:413–423.
- James, C. 2003. Preview: Global Status of Commercialized Transgenic Crops: 2003. ISAAA Briefs No. 30. Ithaca, NY: ISAAA. Web page: <http://www.isaaa.org>. Accessed: 6 June 2005.
- James, C. 2004. Preview: Global Status of Commercialized Biotech/GM Crops: 2004. ISAAA Briefs No. 32. Ithaca, NY: ISAAA. Web page: <http://www.isaaa.org>. Accessed: 6 June 2005.
- Jasieniuk, M., A. L. Brülé-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. Brülé-Babel. 1995. Inheritance of dicamba resistance in wild mustard (*Brassica kaber*). *Weed Sci.* 43:192–195.
- Jordan, N., M. Kelrick, J. Brooks, and W. Kinerk. 1999. Biorational management tactics to select against triazine-resistant *Amaranthus hybridus*: a field trial. *J. Appl. Ecol.* 36:123–132.
- Kaundun, S. S. and J. Windass. 2004. Derived CAPS: a simple method to detect a critical point mutation in the ACCase target gene conferring graminicide resistance in monocot weeds. In Proc. Fourth International Weed Science Congress, Durban, S.A. Davis, CA: International Weed Science Society. P. 50.
- Kemp, M. S., S. R. Moss, and T. H. Thomas. 1990. Herbicide resistance in *Alopecurus myosuroides*. In M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. *Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies*. Washington, DC: American Chemical Society. Pp. 376–393.
- Kremer, E. and L.A.P. Lotz. 1998. Germination and emergence characteristics of triazine-susceptible and triazine-resistant biotypes of *Solanum nigrum*. *J. Appl. Ecol.* 35:302–310.
- Kudsk, P., S. K. Mathiassen, and J. C. Cotterman. 1995. Sulfonylurea resistance in *Stellaria media* [L.] Vill. *Weed Res.* 35:19–24.
- Kulshrestha, G., S. B. Singh, and N. T. Yaduraju. 1999. Mechanism of isoproturon resistance: the metabolism of isoproturon in susceptible and resistant biotypes of *Phalaris minor*. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 167–172.
- LeBaron, H. M. and J. McFarland. 1990. Herbicide resistance in weeds and crops. In M. B. Green, H. M. LeBaron, and W. K. Moberg, eds. *Managing Resistance to Agrochemicals: From Fundamental Research to Practical Strategies*. Washington, DC: American Chemical Society. Pp. 336–352.
- Leeson, J. Y., A. G. Thomas, H. J. Beckie, R. C. Van Acker, and T. Andrews. 2004. Do Manitoba producers reduce in-crop herbicide rates? Proc. 2003 National Mtg. Sainte-Anne-de-Bellevue, QC: Canadian Weed Science Society. P. 89. Web page: <http://www.cwss-scm.ca>. Accessed: 31 May 2005.
- Leeson, J. Y., A. G. Thomas, C. A. Brenzil, and H. J. Beckie. 2006. Do Saskatchewan producers reduce in-crop herbicide rates? Proc. 2004 National Mtg. Sainte-Anne-de-Bellevue, QC: Canadian Weed Science Society. In press.
- Légère, A., H. J. Beckie, F. C. Stevenson, and A. G. Thomas. 2000. Survey of management practices affecting the occurrence of wild oat (*Avena fatua*) resistance to acetyl-CoA carboxylase inhibitors. *Weed Technol.* 14: 366–376.
- Letouzé, A. and J. Gasquez. 1999. A pollen test to detect ACCase target-site resistance within *Alopecurus myosuroides* populations. *Weed Res.* 40: 151–162.
- Letouzé, A. and J. Gasquez. 2001. Inheritance of fenoxaprop-P-ethyl resistance in a blackgrass (*Alopecurus myosuroides* Huds.) population. *Theor. Appl. Genet.* 103:288–296.
- Letouzé, A. and J. Gasquez. 2003. Enhanced activity of several herbicide-degrading enzymes: a suggested mechanism responsible for multiple resistance in blackgrass (*Alopecurus myosuroides* Huds.). *Agronomie* 23: 601–608.
- Li, C., F. C. Yeh, I. N. Morrison, and T. S. Andrews. 2000. Tracing the movement of herbicide resistance in fields infested with *Setaria viridis* L. using amplified fragment length polymorphisms. In A. Légère, ed. Proc. Third International Weed Science Congress, Foz do Iguassu, Brazil. Corvallis, OR: International Weed Science Society. P. 144.
- Little, R. and F. J. Tardif. 2005. Combinations of herbicides at reduced rates for the prevention of herbicide resistance. *Weed Sci. Soc. Am. Abstr.* 45: 111.
- Llewellyn, R. S., R. K. Lindner, D. J. Pannell, and S. B. Powles. 2002. Resistance and the herbicide resource: perceptions of Western Australian grain growers. *Crop Prot.* 21:1067–1075.
- Llewellyn, R. S., R. K. Lindner, D. J. Pannell, and S. B. Powles. 2004. Grain grower perceptions and use of integrated weed management. *Aust. J. Exp. Agric.* 44:993–1001.
- Llewellyn, R. S. and S. B. Powles. 2001. High levels of herbicide resistance

- in rigid ryegrass (*Lolium rigidum*) in the wheat belt of Western Australia. *Weed Technol.* 15:242–248.
- Malik, R. K., G. Gill, and P. R. Hobbs. 1998. Herbicide Resistance—A Major Issue for Sustainable Wheat Productivity in Rice–Wheat Cropping Systems in the Indo-Gangetic Plains. Rice–Wheat Consortium Paper Series 3. New Delhi, India: Rice–Wheat Consortium for the Indo-Gangetic Plains. 36 p.
- Malik, R. K. and S. Singh. 1995. Littleseed canarygrass (*Phalaris minor*) resistance to isoproturon in India. *Weed Technol.* 9:419–425.
- Mallory-Smith, C. A. and E. J. Retzinger, Jr. 2003. Revised classification of herbicides by site of action for weed resistance management strategies. *Weed Technol.* 17:605–619.
- Mallory-Smith, C. A., D. C. Thill, and G. P. Stallings. 1993. Survey and gene flow in acetolactate synthase resistant kochia and Russian thistle. *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 555–558.
- Maneechote, C., C. Preston, and S. B. Powles. 1997. A diclofop-methyl-resistant *Avena sterilis* biotype with a herbicide-resistant acetyl-coenzyme A carboxylase and enhanced metabolism of diclofop-methyl. *Pestic. Sci.* 49:105–114.
- Manley, B. S., H. P. Wilson, and T. E. Hines. 1998. Characterization of imidazolinone-resistant smooth pigweed (*Amaranthus hybridus*). *Weed Technol.* 12:575–584.
- Mansooji, A. M., J. A. Holtum, P. Boutsalis, J. M. Matthews, and S. B. Powles. 1992. Resistance to aryloxyphenoxypropionate herbicides in two wild oat species (*Avena fatua* and *Avena sterilis* ssp. *ludoviciana*). *Weed Sci.* 40:599–605.
- Marshall, R. and S. Moss. 2004. Resistance to acetolactate inhibiting herbicides in UK black-grass (*Alopecurus myosuroides*) populations. *Weed Sci. Soc. Am. Abstr.* 44:15.
- Matthews, J. M. 1994. Management of herbicide resistant weed populations. In S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants—Biology and Biochemistry*. Boca Raton, FL: CRC Press. Pp. 317–335.
- Matthews, J. M. and S. B. Powles. 1992. Aspects of the population dynamics of selection for herbicide resistance in *Lolium rigidum* (Gaud). In J. H. Combellack, K. J. Levick, J. Parsons, and R. G. Richardson, eds. *Proc. First International Weed Control Congress*, Melbourne, Australia. Corvallis, OR: International Weed Science Society. Pp. 318–320.
- Maxwell, B. D. and A. M. Mortimer. 1994. Selection for herbicide resistance. In S. B. Powles and J.A.M. Holtum, eds. *Herbicide Resistance in Plants—Biology and Biochemistry*. Boca Raton, FL: CRC Press. Pp. 1–25.
- Medd, R. W., B. A. Auld, D. R. Kemp, and R. D. Murison. 1987. The influence of wheat density and spatial arrangement on annual ryegrass, *Lolium rigidum* Gaudin, competition. *Aust. J. Agric. Res.* 36:361–371.
- Menendez, J. and R. De Prado. 1996. Diclofop-methyl cross-resistance in a chlorotoluron-resistant biotype of *Alopecurus myosuroides*. *Pestic. Biochem. Physiol.* 56:123–133.
- Monjardino, M., D. J. Pannell, and S. B. Powles. 2003. Multispecies resistance and integrated management: a bioeconomic model for integrated management of rigid ryegrass (*Lolium rigidum*) and wild radish (*Raphanus raphanistrum*). *Weed Sci.* 51:798–809.
- Monjardino, M., D. J. Pannell, and S. B. Powles. 2004. The economic value of haying and green manuring in the integrated management of annual ryegrass and wild radish in a Western Australian farming system. *Aust. J. Exp. Agric.* 44:1195–1203.
- Morrison, I. N. and L. F. Friesen. 1996. Herbicide resistant weeds: mutation, selection, misconception. In H. Brown, G. W. Cussans, M. D. Devine, S. O. Duke, C. Fernandez-Quintanilla, A. Helweg, R. E. Labrada, M. Landes, P. Kudsk, and J. C. Streibig, eds. *Proc. Second International Weed Control Congress*, Copenhagen, Denmark. Flakkebjerg, Slagelse, Denmark: Department of Weed Control and Pesticide Ecology. Pp. 1–9.
- Moss, S. R. 1997. Strategies for the prevention and control of herbicide resistance in annual grass weeds. In R. De Prado, J. Jorrín, and L. García-Torres, eds. *Weed and Crop Resistance to Herbicides*. London: Kluwer Academic. Pp. 283–290.
- Moss, S. R. 2002. Herbicide-resistant weeds. In R.E.L. Naylor, ed. *Weed Management Handbook*. British Crop Protection Council. Oxford, UK: Blackwell Science. Pp. 225–252.
- Moss, S. R., K. M. Cocker, A. C. Brown, L. Hall, and L. M. Field. 2003. Characterisation of target-site resistance to ACCase-inhibiting herbicides in the weed *Alopecurus myosuroides* (black-grass). *Pest Manage. Sci.* 59:190–201.
- Murray, B. G., A. L. Brülé-Babel, and I. N. Morrison. 1996. Two distinct alleles encode for acetyl-CoA carboxylase inhibitor resistance in wild oat (*Avena fatua*). *Weed Sci.* 44:476–481.
- Nazarko, O. M., R. C. Van Acker, and M. H. Entz. 2005. Strategies and tactics for herbicide use reduction in field crops in Canada: A review. *Can. J. Plant Sci.* 85:457–479.
- Neve, P. and S. Powles. 2005a. Recurrent selection with reduced herbicide rates results in the rapid evolution of herbicide resistance in *Lolium rigidum*. *Theor. App. Genet.* 110:1154–1166.
- Neve, P. and S. Powles. 2005b. High survival frequencies at low herbicide use rates in populations of *Lolium rigidum* result in rapid evolution of herbicide resistance. *Heredity* 95:485–492.
- Norsworthy, J. K., J. S. Rutledge, R. E. Talbert, and R. E. Hoagland. 1999a. Agrichemical interactions with propanil on propanil-resistant barnyardgrass (*Echinochloa crus-galli*). *Weed Technol.* 13:296–302.
- Norsworthy, J. K., R. E. Talbert, and R. E. Hoagland. 1999b. Chlorophyll fluorescence evaluation of agrochemical interactions with propanil on propanil-resistant barnyardgrass (*Echinochloa crus-galli*). *Weed Sci.* 47:13–19.
- O'Donovan, J. T., J. C. Newman, R. E. Blackshaw, K. N. Harker, D. A. Derksen, and A. G. Thomas. 1999. Growth, competitiveness, and seed germination of triallate/difenzoquat-susceptible and -resistant wild oat populations. *Can. J. Plant Sci.* 79:303–312.
- Ominski, P. D., M. H. Entz, and N. Kenkel. 1999. Weed suppression by *Medicago sativa* in subsequent cereal crops: a comparative survey. *Weed Sci.* 47:282–290.
- Orson, J. H. 1999. The cost to the farmer of herbicide resistance. *Weed Technol.* 13:607–611.
- Orson, J. H. and D.B.F. Livingston. 1987. Field trials on the efficacy of herbicides on resistant black-grass (*Alopecurus myosuroides*) in different cultivation regimes. *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 887–894.
- Owen, M.D.K. 2001a. Importance of weed population shifts and herbicide resistance in the Midwest USA corn belt. *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 407–412.
- Owen, M.D.K. 2001b. World maize/soybean and herbicide resistance. In S. B. Powles and D. L. Shaner, eds. *Herbicide Resistance and World Grains*. New York: CRC Press. Pp. 101–163.
- Pannell, D. J., V. Stewart, A. Bennett, M. Monjardino, C. Schmidt, and S. B. Powles. 2004. RIM: a bioeconomic model for integrated weed management of *Lolium rigidum* in Western Australia. *Agric. Syst.* 79:305–325.
- Park, K. W., L. Fandrich, and C. A. Mallory-Smith. 2004. Absorption, translocation, and metabolism of propoxycarbazone-sodium in ALS-inhibitor resistant *Bromus tectorum* biotypes. *Pestic. Biochem. Physiol.* 79:18–24.
- Parks, R. J., W. S. Curran, G. W. Roth, N. L. Hartwig, and D. D. Calvin. 1996. Herbicide susceptibility and biological fitness of triazine-resistant and susceptible common lambsquarters (*Chenopodium album*). *Weed Sci.* 44:517–522.
- Pearce, G. A. and J. E. Holmes. 1976. The control of annual ryegrass. *J. Agric. West. Aust.* 17:77–81.
- Pest Management Regulatory Agency (PMRA). 1999. Voluntary Pesticide Resistance-Management Labelling Based on Target Site/Mode of Action. Publ. Regulatory Directive DIR99-06. Ottawa, ON: Health Canada. 24 p. Web page: <http://www.hc-sc.gc.ca/pmra-arla/english/pubs/dir-e.html>. Accessed 21 May 2005.
- Peterson, D. E. 1999. The impact of herbicide-resistant weeds on Kansas agriculture. *Weed Technol.* 13:632–635.
- Poston, D. H., J. Wu, K. K. Hatzios, and H. P. Wilson. 2001. Enhanced sensitivity to cloransulam-methyl in imidazolinone-resistant smooth pigweed. *Weed Sci.* 49:711–716.
- Powles, S. B. 1997. Success from adversity: herbicide resistance can drive changes to sustainable weed management systems. *Proc. Brighton Crop Prot. Conf.—Weeds*. Farnham, UK: British Crop Protection Council. Pp. 1119–1126.
- Powles, S. B. and J. M. Matthews. 1996. Integrated weed management for the control of herbicide-resistant annual ryegrass (*Lolium rigidum*). In H. Brown, G. W. Cussans, M. D. Devine, S. O. Duke, C. Fernandez-Quintanilla, A. Helweg, R. E. Labrada, M. Landes, P. Kudsk, and J. C. Streibig, eds. *Proc. Second International Weed Control Congress*, Co-

- penhagen, Denmark. Flakkebjerg, Slagelse, Denmark: Department of Weed Control and Pesticide Ecology. Pp. 407–414.
- Powles, S. B., M. Monjardino, R. Llewellyn, and D. Pannell. 2000. Proactive versus reactive herbicide resistance management: understanding the economic sense of herbicide conservation versus exploitation. In A. Légère, ed. Proc. Third International Weed Science Congress, Foz do Iguassu, Brazil. Corvallis, OR: International Weed Science Society. Pp. 148–149.
- Powles, S. B., C. Preston, I. B. Bryan, and A. R. Jutsum. 1997. Herbicide resistance: impact and management. *Adv. Agron.* 58:57–93.
- Preston, C. 2004. Herbicide resistance in weeds endowed by enhanced detoxification: complications for management. *Weed Sci.* 52:448–453.
- Preston, C. and C. A. Mallory-Smith. 2001. Biochemical mechanisms, inheritance, and molecular genetics of herbicide resistance in weeds. In S. B. Powles and D. L. Shaner, eds. *Herbicide Resistance and World Grains*. New York: CRC Press. Pp. 23–60.
- Preston, C. and S. B. Powles. 2002a. Evolution of herbicide resistance in weeds: initial frequency of target site-based resistance to acetolactate synthase-inhibiting herbicides in *Lolium rigidum*. *Heredity* 88:8–13.
- Preston, C. and S. B. Powles. 2002b. Mechanisms of multiple herbicide resistance in *Lolium rigidum*. In J. M. Clark and I. Yamaguchi, eds. *Agronomical Resistance: Extent, Mechanism, and Detection*. ACS Symposium Series No. 808. Washington, DC: American Chemical Society. Pp. 150–160.
- Preston, C., F. J. Tardif, J. T. Christopher, and S. B. Powles. 1996. Multiple resistance to dissimilar herbicide chemistries in a biotype of *Lolium rigidum* due to enhanced activity of several herbicide degrading enzymes. *Pestic. Biochem. Physiol.* 54:123–134.
- Putwain, P. D. 1982. Herbicide resistance in weeds—an inevitable consequence of herbicide use? Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 719–728.
- Radosevich, S., J. Holt, and C. Ghersa. 1997. Genetics and evolution of weeds. In S. Radosevich, J. Holt, and C. Ghersa, eds. *Weed Ecology: Implications for Management*. 2nd ed. New York: Wiley. Pp. 69–102.
- Riches, C. R., J. S. Knights, L. Chaves, J. C. Caseley, and B. E. Valverde. 1997. The role of pendimethalin in the integrated management of propanil-resistant *Echinochloa colona* in Central America. *Pestic. Sci.* 51: 341–346.
- Richter, J. and S. B. Powles. 1993. Pollen expression of herbicide target site resistance genes in annual ryegrass (*Lolium rigidum*). *Plant Physiol.* 102: 1037–1041.
- Rieger, M. A., L. Stone, and C. Preston. 2001. Herbicide resistant gene movement on a landscape scale with *Lactuca serriola*. *Weed Sci. Soc. Am. Abstr.* 41:125.
- Ritter, R. L. and H. Menbere. 1997. Distribution and management of triazine-resistant weeds in the Mid-Atlantic region of the U.S.A. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 1147–1152.
- Rotteveel, T.J.W., J.W.F.M. de Goeij, and A. F. van Gemerden. 1997. Towards the construction of a resistance risk evaluation scheme. *Pestic. Sci.* 51: 407–411.
- Rubin, B. 1991. Herbicide resistance in weeds and crops, progress and prospects. In J. C. Caseley, G. W. Cussans, and R. K. Atkin, eds. *Herbicide Resistance in Weeds and Crops*. Oxford, U.K.: Butterworth-Heinemann. Pp. 387–414.
- 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* L.) is controlled by a single nuclear recessive gene. *J. Hered.* 94:523–527.
- Sattin, M., M. A. Gasparetto, and C. Campagna. 2001. Situation and management of *Avena sterilis* ssp *ludoviciana* and *Phalaris paradoxa* resistant to ACCase inhibitors in Italy. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 755–762.
- Schmidt, L. A., R. E. Talbert, and M. McClelland. 2004. Management of acetolactate synthase (ALS)-resistant common cocklebur (*Xanthium strumarium*) in soybean. *Weed Technol.* 18:665–674.
- Seefeldt, S. S., D. R. Gealy, B. D. Brewster, and E. P. Fuerst. 1994. Cross-resistance of several diclofop-resistant wild oat (*Avena fatua*) biotypes from the Willamette Valley of Oregon. *Weed Sci.* 42:430–437.
- Seefeldt, S. S., R. Zemetra, F. L. Young, and S. S. Jones. 1998. Production of herbicide-resistant jointed goatgrass (*Aegilops cylindrica*) × wheat (*Triticum aestivum*) hybrids in the field by natural hybridization. *Weed Sci.* 46:632–634.
- Shaner, D. L., D. A. Feist, and E. J. Retzinger. 1997. SAMOA: one company's approach to herbicide-resistant weed management. *Pestic. Sci.* 51:367–370.
- Shaner, D. L., S. Howard, and I. Chalmers. 1999. Effectiveness of mode of action labelling for resistance management: a survey of Australian farmers. Proc. Brighton Crop Prot. Conf.—Weeds. Farnham, UK: British Crop Protection Council. Pp. 797–802.
- Shirliffe, S. J. and M. H. Entz. 2005. Chaff collection reduces seed dispersal of wild oat (*Avena fatua*) by a combine harvester. *Weed Sci.* 53:465–470.
- Sibony, M. and B. Rubin. 2003. Molecular basis for multiple resistance to acetolactate synthase-inhibiting herbicides and atrazine in *Amaranthus blitoides* (prostrate pigweed). *Planta* 216:1022–1027.
- Siminszky, B., N. P. Coleman, and M. Naveed. 2005. Denaturing high-performance liquid chromatography efficiently detects mutations of the acetolactate synthase gene. *Weed Sci.* 53:146–152.
- Singh, S., R. C. Kirkwood, and G. Marshall. 1998. Effect of the monooxygenase inhibitor piperonyl butoxide on the herbicidal activity and metabolism of isoproturon in herbicide resistant and susceptible biotypes of *Phalaris minor* and wheat. *Pestic. Biochem. Physiol.* 59:143–153.
- Singh, S., R. C. Kirkwood, and G. Marshall. 1999. Biology and control of *Phalaris minor* Retz. (littleseed canarygrass) in wheat. *Crop Prot.* 18:1–16.
- Smeda, R. J., R. S. Currie, and J. H. Rippee. 2000. Fluazifop-P resistance expressed as a dominant trait in sorghum (*Sorghum bicolor*). *Weed Technol.* 14:397–401.
- Sprague, C. L., E. W. Stoller, and L. M. Wax. 1997. Response of an acetolactate synthase (ALS)-resistant biotype of *Amaranthus rudis* to selected ALS-inhibiting and alternative herbicides. *Weed Res.* 37:93–101.
- Stankiewicz, M., G. Gadamski, and S. W. Gawronski. 2001. Genetic variation and phylogenetic relationships of triazine-resistant and triazine-susceptible biotypes of *Solanum nigrum*—analysis using RAPD markers. *Weed Res.* 41:287–300.
- Steckel, L., B. Hayes, T. Mueller, K. Smith, S. Nichols, A. Kendig, C. Craig, and B. Nichols. 2005. Impact of glyphosate-resistant horseweed on conservation tillage in the North Delta region. *Weed Sci. Soc. Am. Abstr.* 45:42.
- Stephenson, G. R., M. D. Dykstra, R. D. McLaren, and A. S. Hamill. 1990. Agronomic practices influencing triazine-resistant weed distribution in Ontario. *Weed Technol.* 4:199–207.
- Sterling, T. M., I. Ray, A. D. Vallotton, and R. P. Sabba. 2002. Recessive inheritance of picloram resistance in yellow starthistle (*Centaurea solstitialis* L.). *Weed Sci. Soc. Am. Abstr.* 42:11.
- Talbert, R. E., L. A. Schmidt, J. S. Rutledge, E. E. Scherder, M. L. Lovelace, N. W. Buehring, and F. L. Baldwin. 2000. Alternative herbicide programs for control of propanil-resistant *Echinochloa crus-galli* (L.) Beauv. in drill-seeded rice. In A. Légère, ed. Proc. Third International Weed Science Congress, Foz do Iguassu, Brazil. Corvallis, OR: Inter. Weed Sci. Soc. P. 149.
- Tardif, F. J. and S. B. Powles. 1999. Effect of malathion on resistance to soil-applied herbicides in a population of rigid ryegrass (*Lolium rigidum*). *Weed Sci.* 47:258–261.
- Thill, D. C. and D. Lemerle. 2001. World wheat and herbicide resistance. In S. B. Powles and D. L. Shaner, eds. *Herbicide Resistance and World Grains*. New York: CRC Press. Pp. 165–194.
- Thill, D. C., J. T. O'Donovan, and C. A. Mallory-Smith. 1994. Integrated weed management strategies for delaying herbicide resistance in wild oats. *Phytoprotection* 75(Suppl.):61–70.
- Thomas, A. G., J. Y. Leeson, H. J. Beckie, and L. M. Hall. 2003. Herbicide use patterns and herbicide-resistant weed awareness: results from grower surveys in Alberta, Canada. *Weed Sci. Soc. Am. Abstr.* 43:66.
- Tsuji, R., A. J. Fischer, M. Yoshino, A. Roel, J. E. Hill, and Y. Yamasue. 2003. Herbicide-resistant late watergrass (*Echinochloa phyllopogon*): similarity in morphological and amplified fragment length polymorphism traits. *Weed Sci.* 51:740–747.
- United States (U.S.) Environmental Protection Agency. 2001. Pesticide Registration (PR) Notice 2001–5. EPA 730-N-01-005. Washington, DC. 30 p.
- Valverde, B. E. 1996. Management of herbicide resistant weeds in Latin America: the case of propanil-resistant *Echinochloa colona* in rice. In H. Brown, G. W. Cussans, M. D. Devine, S. O. Duke, C. Fernandez-Quintanilla, A. Helweg, R. E. Labrada, M. Landes, P. Kudsk, and J. C. Streibig, eds. Proc. Second International Weed Control Congress, Copenhagen, Denmark. Flakkebjerg, Slagelse, Denmark: Department of Weed Control and Pesticide Ecology. Pp. 415–420.

- Valverde, B. E., L. Chaves, I. Garita, F. Ramírez, E. Vargas, J. Carmiol, C. R. Riches, and J. C. Casely. 2001. Modified herbicide regimes for propanil-resistant junglerice control in rain-fed rice. *Weed Sci.* 49:395–405.
- Valverde, B. E., I. Garita, E. Vargas, L. Chaves, F. Ramirez, A. J. Fischer, and H. Pabon. 1999. Anilofos as a synergist to propanil for controlling propanil-resistant junglerice, *Echinochloa colona*. *Weed Sci. Soc. Am. Abstr.* 39:159.
- Valverde, B. E., C. R. Riches, and J. C. Caseley. 2000. Prevention and Management of Herbicide Resistant Weeds in Rice: Experiences from Central America with *Echinochloa colona*. San José, Costa Rica: Cámara de Insumos Agropecuarios. 123 p.
- VanGessel, M. J. 2001. Glyphosate-resistant horseweed from Delaware. *Weed Sci.* 49:703–705.
- Veldhuis, L. J., L. M. Hall, J. T. O'Donovan, W. Dyer, and J. C. Hall. 2000. Metabolism-based resistance of a wild mustard (*Sinapis arvensis* L.) biotype to ethametsulfuron-methyl. *J. Agric. Food Chem.* 48:2986–2990.
- Vila-Aiub, M. M., P. Neve., K. J. Steadman, and S. B. Powles. 2005. Ecological fitness of a multiple herbicide-resistant *Lolium rigidum* population: dynamics of seed germination and seedling emergence of resistant and susceptible phenotypes. *J. Appl. Ecol.* 42:288–298.
- Warwick, S. I., H. J. Beckie, M. J. Simard, A. Légère, H. Nair, and G. Séguin-Swartz. 2004. Environmental and agronomic consequences of herbicide-resistant (HR) canola in Canada. In H.C.M. den Nijs, D. Bartsch, and J. Sweet, eds. *Introgression from Genetically Modified Plants (GMP) into Wild Relatives*. Wallingford, Oxfordshire, UK: CABI Publ. Pp. 323–337.
- Warwick, S. I., H. J. Beckie, and E. Small. 1999. Transgenic crops: new weed problems for Canada? *Phytoprotection* 80:71–84.
- Warwick, S. I., M. J. Simard, A. Légère, H. J. Beckie, L. Braun, B. Zhu, P. Mason, G. Séguin-Swartz, and C. N. Stewart. 2003. Hybridization between transgenic *Brassica napus* L. and its wild relatives: *Brassica rapa* L., *Raphanus raphanistrum* L., *Sinapis arvensis* L., and *Erucastrum gallicum* (Willd.) O. E. Schulz. *Theor. Appl. Genet.* 107:528–539.
- Weersink, A., R. S. Llewellyn, and D. J. Pannell. 2005. Economics of preemptive management to avoid weed resistance to glyphosate in Australia. *Crop Prot.* 24:659–665.
- Werck-Reichhart, D., A. Hehn, and L. Didierjean. 2000. Cytochromes P450 for engineering herbicide tolerance. *Trends Plant Sci.* 5:116–123.
- Westra, P., S. Nissen, and D. Karam. 2000. Risk factors that impact resistant weed selection. In A. Légère, ed. *Proc. Third International Weed Science Congress, Foz do Iguassu, Brazil*. Corvallis, OR: International Weed Science Society. Pp. 154–155.
- Willis, A. D., A. M. Mortimer, P. D. Putwain, and S. R. Moss. 1997. Half-sib analysis of the genetic basis of graminicide resistance in blackgrass, *Alopecurus myosuroides* L. (Huds.) in the UK. *Integrated Approach to Combating Resistance: Resistance '97 Abstracts*. Harpenden, Herts, UK: IACR-Rothamsted.
- 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.
- Zemetra, R., J. Hansen, and C. A. Mallory-Smith. 1998. Potential for gene transfer between wheat (*Triticum aestivum*) and jointed goatgrass (*Aegilops cylindrica*). *Weed Sci.* 46:313–317.
- Zhang, J., S. E. Weaver, and A. S. Hamill. 2000. Risks and reliability of using herbicides at below-labeled rates. *Weed Technol.* 14:106–115.