
Current State of Herbicide Resistance in Canada

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Abstract

Biotypes of wild oat (*Avena fatua* L.) resistant to Group 1 herbicides in the prairie provinces, and biotypes of chickweed [*Stellaria media* (L.) Vill.] and kochia [*Kochia scoparia* (L.) Schrad.] in western Canada and pigweed (*Amaranthus*) species in Ontario resistant to Group 2 herbicides are most abundant and widespread. Evolution of resistance in these biotypes is attributable to frequent use of herbicides from these two respective groups. Increasing incidence of wild oat populations with multiple-group resistance will threaten the future effectiveness of herbicides of different modes of action. Proactive or reactive management for herbicide resistance in weeds must consider the risks of herbicides of different modes of action to select for resistance and the differing propensity of herbicides to be metabolized in herbicide-resistant biotypes when rotating among herbicides, must meet criteria for effective herbicide mixtures, and should incorporate agronomic practices in cropping systems that help reduce weed seed production and spread.

Introduction

To date, 249 herbicide-resistant (HR) weed biotypes (156 species) have been reported in 47 countries; 35 biotypes are in Canada (Heap 2001). Crop monoculture and reliance on the same herbicide group have been implicated in most cases. During the past 30 years, HR weeds have been managed successfully by producers worldwide, primarily through the use of alternative herbicides. Important exceptions are multiple-group HR grass species that are controlled by few or no alternative herbicides. The purpose of this paper is to summarize HR weed biotypes in Canada, identify herbicide-use patterns and crop rotations that have influenced the selection of herbicide resistance, and assess the future outlook for preventing and managing HR weeds.

Group 1 Resistance

Group 1 herbicides, which include two chemical families, the aryloxyphenoxy propionates (APP) and cyclohexanediones (CHD), have been widely used since their introduction in the late 1970s and early 1980s. One-half or more of the sprayed acreage across the prairies received a Group 1 herbicide application in the mid-to-late 1990s (Beckie et al. 1999c). Surveys conducted across the prairies in 1996 and 1997 indicated that wild oat resistance to Group 1 herbicides occurred most frequently relative to other herbicide groups (Beckie et al. 1999c, 2001b). Frequency of Group 1-HR wild oat was directly related to frequency of Group 1 herbicide use. Group 1-HR wild oat occurred in approximately one-half of fields surveyed in each of the three

prairie provinces. A field survey conducted in 1996 and a grain elevator survey conducted in 1997 determined that 20% of fields and 83% of elevators had Group 1-HR green foxtail [*Setaria viridis* (L.) Beauv.] (Beckie et al. 1999b). Crop rotations had little influence on use patterns, because Group 1 herbicides are registered for use in cereal, oilseed, and pulse crops (Légère et al. 2000). Resistance to Group 1 herbicides typically develops after six to ten applications.

Incidence of APP resistance in wild oat and green foxtail is markedly higher than CHD resistance (Beckie et al. 1999b, c, 2001b). However, cross-resistance patterns may not be predicted from herbicide-use patterns. In a risk-assessment study, resistance to CHD herbicides was not related to CHD use but to frequency of Group 1 herbicide use (i.e., CHD plus APP), suggesting that the pressure imposed by APPs contributed to the selection of CHD resistance in wild oat (Légère et al. 2000). The presence of patches with different cross-resistance patterns within a field (Andrews et al. 1998; Bourgeois et al. 1997a) illustrates the difficulty in delaying or managing Group 1-HR wild oat by alternating APPs and CHDs in a field.

As the frequency of occurrence of Group 1 resistance increases, there are more opportunities for gene flow to influence occurrence. Wild oat is primarily self-pollinating (outcrossing up to 12%) and green foxtail is highly selfing. Hence, the spread of HR wild oat and green foxtail is more likely due to seed spread via machinery or non-composted manure. Wild oat can spread greater than 150 m by a combine harvester (Shirtliffe et al. 1998). Seed spread of HR wild oat and green foxtail within and among fields has been documented (Andrews et al. 1998; Li et al. 2000). Management practices that limit the spread of HR seed can slow the occurrence of resistance. In the 1996 field survey in Saskatchewan, producers who practiced weed sanitation were less likely to have HR wild oat than those who were less careful (Légère et al. 2000).

Group 2 Resistance

Even though Group 2 products were introduced relatively recently (1982), the greatest number of weed biotypes worldwide are resistant to this herbicide group (Heap 2001). Similar to Group 1 resistance, patterns of cross-resistance cannot be predicted based on field histories and must be assessed for each population.

In western Canada, Group 2 resistance has been documented in eight broadleaf weed species: multiple populations of chickweed since 1988 in central Alberta (Morrison and Devine 1994; O'Donovan et al. 1994a), a biotype of Russian thistle [*Salsola pestifer* (A.) Nels.] in 1989 in Saskatchewan (Morrison and Devine 1994), a biotype of common hempnettle (*Galeopsis tetrahit* L.) in 1995 in Manitoba (Heap 2001), a biotype of false cleavers (*Galium spurium* L.) in 1996 in Alberta (Hall et al. 1998), two biotypes of spiny annual sow thistle [*Sonchus asper* (L.) Hill] in 1996 and a ball mustard (*Neslia paniculata* L.) biotype in 1998 in Alberta (Heap 2001), and a biotype of wild mustard (*Sinapis arvensis* L.) in 1992 in Manitoba and in 1993 in Alberta (Heap 2001; Jeffers et al. 1996). In the latter HR biotype, resistance was attributed to enhanced metabolism (Hall et al. 1999; Veldhuis et al. 2000). This was the first report of metabolism-based resistance in a broadleaf weed to a sulfonyleurea herbicide. Kochia HR biotypes have been reported since 1988 in the three prairie provinces (Morrison and Devine 1994). Over 50 populations of Group 2-HR kochia have been documented in southern Saskatchewan and

southern Alberta during the past five years. Gene flow through pollen and seed movement markedly aids in the dispersal of resistance in this species.

Patterns of herbicide use have contributed to the selection for Group 2 resistance in broadleaf weeds. Resistance in broadleaf weeds typically develops after four to seven applications. Group 2 herbicide use is higher in Alberta and Manitoba than in Saskatchewan (Figure 1).

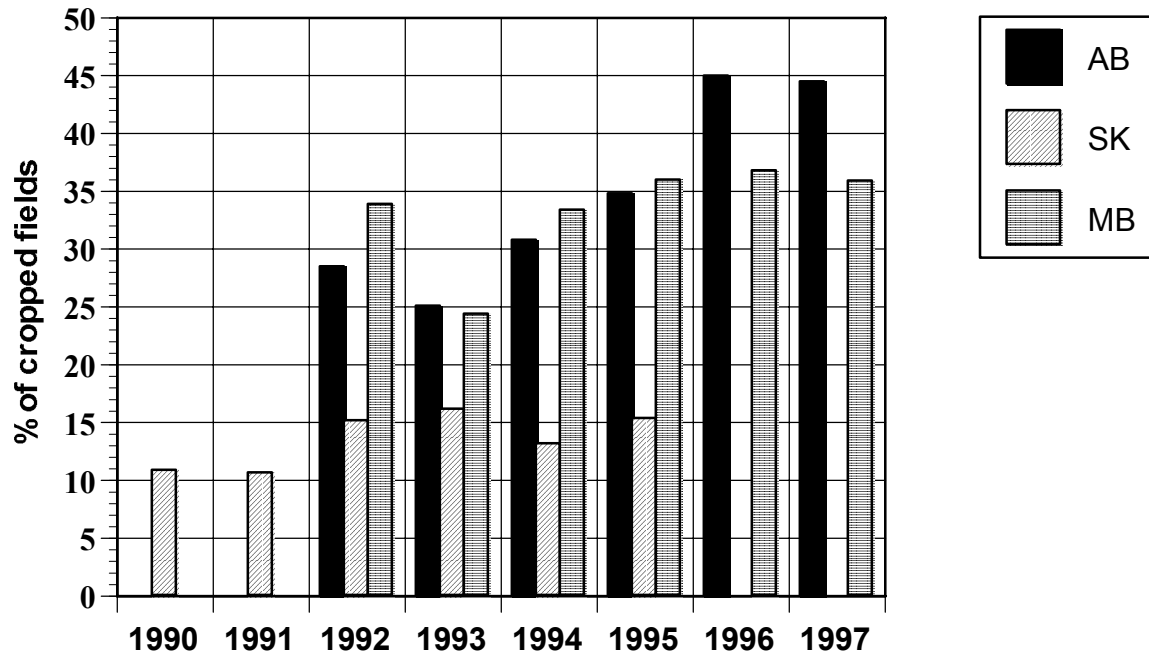


Figure 1. Group 2 herbicide use in the prairies (Source: A. G. Thomas).

In Ontario, resistance was confirmed in nine green pigweed (Powell amaranth) [*Amaranthus powellii* (S.) Wats.] populations and four redroot pigweed (*Amaranthus retroflexus* L.) populations collected in southwestern Ontario in 1997 (Ferguson et al. 2000b). Furthermore, cross-resistance to thifensulfuron was found in two populations of green pigweed and two populations of redroot pigweed. Selection occurred primarily in soybean (*Glycine max* L.) where reliance on Group 2 herbicides had been fairly high in the 1990s. Imazethapyr dominated the soybean market for most of the 1990s. In 1997, more than 75% of the soybean crop in Ontario was treated with at least one Group 2 herbicide.

In grass weeds, Group 2 resistance has been reported in wild oat. In a survey of fields in two randomly selected townships in 1997 (Beckie et al. 2001b), 20 to 30% of fields had wild oat populations exhibiting Group 2 resistance. In another survey in the province that year, 23% of grain elevators had Group 2-HR wild oat (Beckie et al. 1999c). In Manitoba in 1997, 21% of fields sprayed with imazamethabenz had Group 2-HR wild oat (Beckie et al. 1999c).

Group 3 Resistance

Despite extensive and sustained use in western Canada, only a few biotypes with Group 3 resistance have been reported worldwide. Group 3 resistance in green foxtail, discovered in 1988

in Manitoba, typically developed after 15 to 20 applications. The persistence of trifluralin resistance between 1988 and 1995 in fields infested with HR green foxtail suggests no apparent fitness penalty (Andrews and Morrison 1997). In southwestern Manitoba, one in four fields is estimated to have Group 3-HR green foxtail (Goodwin 1994). In a field survey in Saskatchewan in 1996, 11% of fields had Group 3-HR green foxtail; two-thirds of these fields were located in the Parkland region where frequency of fields with this weed species is highest (Beckie et al. 1999b). In contrast to green foxtail, incidence of Group 3-HR biotypes of wild oat is rare. No resistance was detected in populations in high-risk fields in Saskatchewan in 1996.

Group 4 Resistance

Although auxinic herbicides have been used frequently since 1946 (e.g., Figure 2), there are relatively few reports of resistance to these compounds. Resistance typically evolves after 15 to 30 applications. In 1990, biotypes of wild mustard resistant to dicamba, 2,4-D, MCPA, dichlorprop, mecoprop, and picloram were discovered in west-central Manitoba, after selection with a mixture of MCPA, mecoprop, and dicamba for 10 consecutive years in addition to auxinic herbicides used previously (Heap and Morrison 1992). Resistance was conferred by a single, dominant nuclear allele (Jasieniuk et al. 1995). This simple inheritance, which facilitates rapid resistance evolution, was not expected because of the low incidence of HR biotypes to this mode of action despite long-term and widespread use of these herbicides. In 1998 in Alberta, a common hempnettle biotype resistant to dicamba and MCPA was reported (Heap 2001).

Wild carrot (*Daucus carota* L.) with resistance to 2,4-D within 5 years of continuous use, was discovered along a roadside near Milton, Ontario in 1957 (Switzer 1957). This occurrence was one of the first cases of herbicide resistance. In 1998, seeds were collected along the roadside and the persistence of resistance in the population was confirmed (Van Eerd et al. 2000).

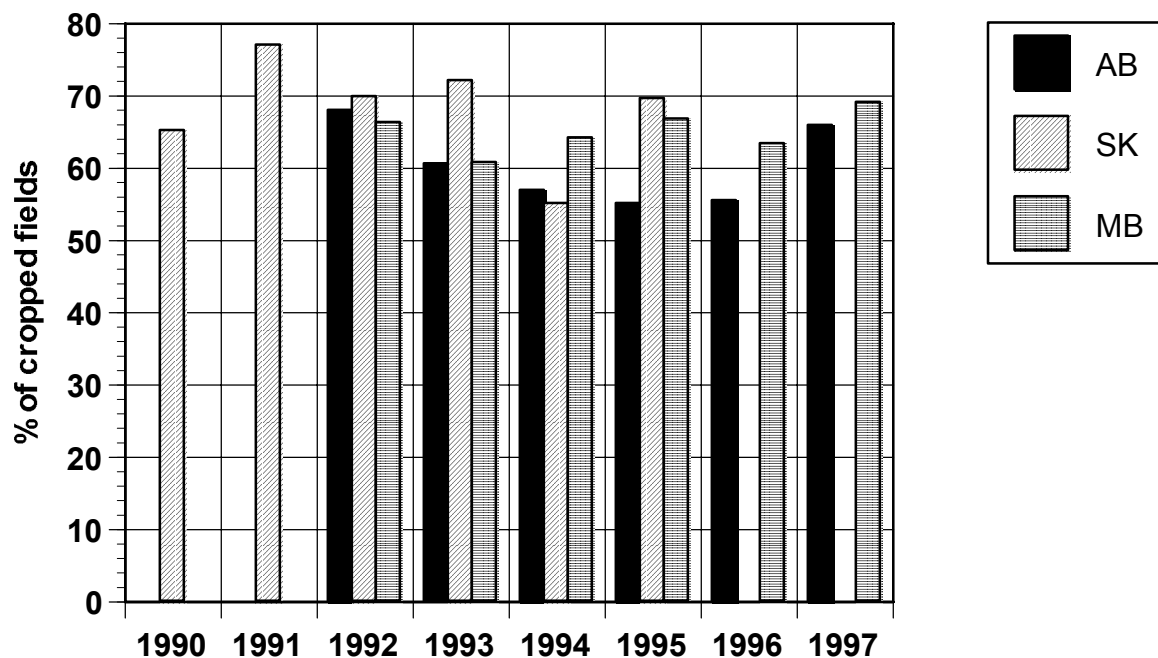


Figure 2. Group 4 herbicide use in the prairies (Source: A. G. Thomas).

Group 5 Resistance

In western Canada, a common groundsel (*Senecio vulgaris* L.) population resistant to triazine herbicides was reported in British Columbia in 1978. In 1994, a metribuzin-HR biotype of wild mustard was found in a field in southern Manitoba where metribuzin had been applied frequently (L. F. Friesen, personal communication).

The greatest number of weed biotypes in eastern Canada are resistant to the triazines. Resistance to the triazines was first documented in common lambsquarters (*Chenopodium album* L.) in 1974 in Ontario. Since then, a total of 10 species have evolved triazine-HR biotypes in the province. Resistance to triazines has also been found in Quebec and in the Maritime region. Of all the species in Ontario, common lambsquarters and *Amaranthus* species have spread the most, causing the most concern to producers. HR biotypes generally have a high level of resistance to the triazines and moderate resistance to triazinones, but little or no resistance to phenylureas.

Triazine resistance was an issue of greater importance in the 1980s than now. At that time, corn (*Zea mays* L.) was grown as a monoculture with high rates of atrazine and little tank mixing with other broadleaf weed herbicides. Triazine resistance is no longer a major preoccupation for producers. The reliance on atrazine has been reduced due to the prevalence of soybean as a rotational crop with corn and the availability of alternative herbicides. Atrazine is still being used, but seldom alone and at much lower rates than in previous decades. Thus, selection pressure has been reduced and HR biotypes are being controlled successfully.

Group 7 Resistance

This group of herbicides, of which linuron is the most widely used in eastern Canada, has a similar mode of action as the triazines, but has a slightly different binding site and resistance is relatively rare compared to the triazines. Linuron is registered for use in a range of crops such as corn, edible bean (*Phaseolus vulgaris* L.), soybean, carrot, etc. Its use in field crops is now relatively limited, but in carrots it is one of the few broadleaf weed herbicides available.

Two distinct cases of linuron-selected resistance have recently been reported in eastern Canada from fields that were in carrot production. Common ragweed (*Ambrosia artemisiifolia* L.) biotypes from southwestern Quebec have been reported to have developed linuron resistance (Saint-Louis et al. 2000). A biotype of green pigweed originating from a carrot field in Ontario is resistant to linuron and has cross-resistance to some triazine herbicides. The number of fields affected as well as the spread of this biotype is unknown, but it would be expected that resistance is limited to fields where linuron was heavily used.

Group 8 Resistance

In a long-term study, resistance in wild oat occurred after 18 years where triallate was applied annually in continuous wheat, but not where triallate was applied 10 times in a wheat-fallow rotation over the same period (Beckie and Jana 2000), similar to the rate of evolution noted by O'Donovan et al. (1994b). Triallate-HR biotypes are also resistant to the chemically unrelated herbicide difenzoquat, even though little history of use in infested fields was evident. Most of

these fields were under monoculture barley or wheat production. About 15% of fields and 24% of grain elevators in Saskatchewan in 1997 and 19% of fields in Manitoba had Group 8-HR wild oat (Beckie et al. 1999c, 2001b).

There is little difference in fitness between HR and susceptible (HS) biotypes (O'Donovan et al. 1999). However, seeds from HR populations are less dormant than those from HS populations, which may at least partially explain a general decline observed in the level of HR:HS wild oat from 1990 to 1997 in fields in Alberta (O'Donovan et al. 2000). However, most fields still contained high levels of HR wild oat, especially where triallate or difenzoquat was applied more than twice during 1988 and 1997. Greater and more rapid emergence of HR individuals compared to HS individuals, analogous to that of Group 2-HR kochia biotypes, may be potentially exploited for selective HR biotype control prior to seeding.

Group 9 Resistance

Glyphosate was commercially introduced in 1974 and is now one of the world's most widely used herbicide. A survey of 53 no-till fields at high risk for glyphosate resistance (10 to 24 years of consecutive use) in the three prairie provinces in 1998 failed to detect glyphosate-HR biotypes.

Group 22 Resistance

Resistance to paraquat has been documented in horseweed [*Conyza canadensis* (L.) Cronq.] and Virginia pepperweed (*Lepidium virginicum* L.) (Smisek et al. 1998). These HR biotypes occurred in fruit orchards in Essex County, Ontario where paraquat was used intensively (3 to 5 times a year for at least 10 years) to control weeds between trees. Because it appears that the populations have not spread from the affected orchards, resistance is contained. Producers have simply switched to using glyphosate to manage these HR populations.

Multiple-Group Resistance

Three wild oat populations in northwestern Manitoba in 1994 were discovered to be resistant to fenoxaprop-P (Group 1), imazamethabenz (Group 2), and flamprop (Group 25) (Friesen et al. 2000; Morrison et al. 1995). In a field survey in Saskatchewan in 1996, 20% of Group 1-HR populations were also resistant to Group 2 herbicides, even though these herbicides were not frequently used (Beckie et al. 1999c). In a survey of two randomly selected townships in Saskatchewan in 1997, multiple-group resistance (1,2; 1,8; 2,8; 1,2,8) were exhibited in wild oat populations in 30 to 40% of fields in both townships (Beckie et al. 2001b). In Manitoba in 1997, 27% of fields surveyed had wild oat resistant to herbicides from more than one group; four populations were resistant to all herbicides registered for use in wheat (Groups 1, 2, 8, 25) (Beckie et al. 1999c). Similar to the multiple-group populations discovered in 1994, the fields had a history of Group 1 herbicide use only. An additional five quadruple-group HR populations from northwestern Manitoba have since been confirmed.

Multiple-group HR wild oat biotypes are conferred either by a single mechanism (e.g. enhanced metabolism), or multiple mechanisms selected sequentially with herbicides of different modes of

action. Metabolism-based resistance is more probable where herbicide-use histories indicate little or no use of herbicides from one or more herbicide groups to which the biotype exhibits resistance. For example, the likely mechanism conferring multiple-group resistance in the wild oat biotypes from northwestern Manitoba is enhanced metabolism (Friesen and Hall 2000). Metabolic resistance appears more frequently in monocot weeds and crops than in dicot plants. Non-selective herbicides do not select for metabolism-based resistance. For example, quizalofop, which controls grass weeds in dicot but not monocot crops, selects only for target site resistance, whereas fenoxaprop, which is used in both monocot and dicot crops, selects for both target site and metabolism-based resistance. Herbicides that are detoxified via pathways different than that mediated by cytochrome P450 oxygenases or that are not metabolized will lessen the chance of selecting for multiple-group (metabolism-based) HR grass weed populations.

Group 1- and 3-HR green foxtail in Manitoba (Heap and Morrison 1996) and Saskatchewan (Beckie et al. 1999b) is likely due to two resistance mechanisms within individuals. HR biotypes were initially selected with Group 3 products; control of these HR biotypes with Group 1 herbicides selected for multiple-group HR biotypes. Similarly, resistance to Group 2 herbicides and to the Group 4 herbicide, quinclorac, in a biotype of false cleavers is likely due to two mechanisms. Group 2 resistance in this biotype is due to target site insensitivity, whereas the mechanism of quinclorac resistance is unknown (Hall et al. 1998). One green pigweed population with triazine (Group 5) and imazethapyr (Group 2) resistance has been discovered in Ontario (Ferguson et al. 2000a). High level resistance to atrazine was confirmed, with a lower level of resistance to metribuzin. Treatment of this population with mixtures of imazethapyr plus atrazine or imazethapyr plus metribuzin had no visible affect on HR individuals.

Outlook for Delaying or Managing Herbicide Resistance

Around the world, producers have been reluctant to proactively manage weeds to delay the selection for herbicide resistance. If the cost and effort of prevention is the same as that of the cure, producers are reluctant to change their program until after resistance has occurred. Selection pressure has the greatest impact on resistance development and is a factor that producers can reduce or vary by the use of herbicide rotations, mixtures, or application timing. Producers are increasingly practicing herbicide group rotation (Beckie et al. 1999a; Bourgeois et al. 1997b; Goodwin 1994). Herbicide rotations or mixtures generally have the greatest effect in delaying resistance when the mechanism conferring resistance is target site-based, target weed species are highly self-pollinated, and seed spread is restricted. For example, there is little Group 2 resistance in Europe or Japan where Group 2 herbicides are used in rotation or mixture with other herbicides. If mixing partners do not meet the criteria of similar persistence and efficacy and different propensity for selecting for resistance in target species, the effectiveness of mixtures for delaying resistance will be reduced and may inadvertently accelerate evolution of multiple resistance. Metabolism-based resistance conferring resistance to herbicides with different modes of action will clearly limit the effectiveness of herbicide group rotation as a tool to delay resistance. Guidelines for rotating herbicides with different propensity to be metabolized need to be developed to combat increasing cases of metabolism-based HR grass weed populations.

Not all herbicides have the same probability of selecting for resistance in weeds (Figure 3). The ‘one in three’ rule advocated in the 1990s pertained to all herbicides, regardless of mode of action. Although it was a good rule of thumb for herbicide group rotation, it was based on anecdotal evidence for time of evolution of Group 1 resistance in wild oat. Producers need more sophisticated information now. Simply stated, the higher the risk of a herbicide mode of action of selecting for resistance, the less often herbicides from that group should be applied. It is widely agreed that Group 1 and 2 herbicides pose a high risk for selecting HR biotypes relative to herbicides from other groups. Lower risk herbicides should be used to reduce the number of weeds selected with higher risk herbicides.

More information provided to producers to tailor label rates according to environment conditions and herbicide sensitivity of the species present in their fields is required to avoid over-dosing. For example, reduced but effective Group 2 herbicide rates used to control chickweed in Europe compared to those used in western Canada halved the time for resistance.

With the exception of frequency of fallow in the rotation and sanitation practices, resistance development in wild oat was little affected by cultural practices used by producers (Légère et al. 2000). Non-chemical weed management practices are recommended to reduce herbicide inputs, but they will not affect the selection pressure if herbicide use patterns in a field remain the same.

Multiple-group resistance has been the chief impetus for the adoption of integrated weed management (IWM) strategies worldwide. Based on past global experience, producers only adopt IWM strategies after the development of resistance, thus managing resistance retroactively not proactively. In Canada, the increasing size of farms with concomitant limited labor and time availability have reinforced our heavy reliance on herbicides as the predominant weed control tool. This trend will not decline significantly in the near future. However, there is ample room for using herbicides smarter. Varied and reduced selection pressure is key in achieving this goal.

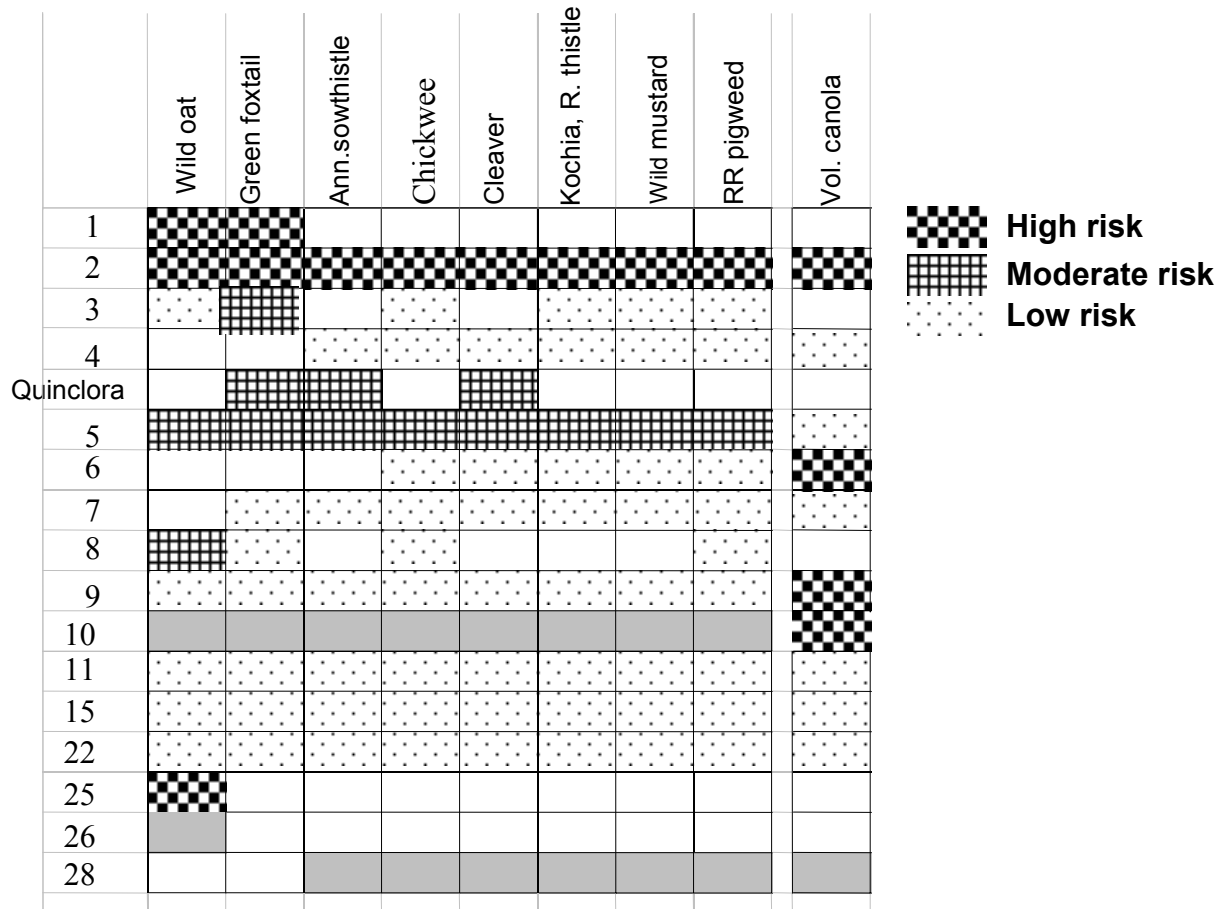


Figure 3. Relative risk of herbicides for selecting for resistance in various weeds: high risk: ≤ 10 applications; low risk: > 20 applications [gray fill: insufficient data; no fill: not controlled] (Beckie et al. 2001a).

Literature Cited

- 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.
- 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., L. M. Hall, and F. J. Tardif. 2001a. Herbicide resistance in Canada – where are we today? Proc. Integrated Weed Management Symposium, Expert Committee on Weeds, Banff, AB. Nov. 2000 (*in press*).
- Beckie, H. J. and S. Jana. 2000. Selecting for triallate resistance in wild oat. *Can. J. Plant Sci.* 80:665-667.
- 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. 2001b. Survey of herbicide-resistant wild oat (*Avena fatua*) in two townships in Saskatchewan. *Can. J. Plant Sci.* (submitted).
- 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 producer survey of ACCase resistant wild oat in Manitoba. *Can. J. Plant Sci.* 77:709-715.
- Ferguson, G. M., R. S. Diebold, F. J. Tardif, and A. S. Hamill. 2000a. Populations of green pigweed (*Amaranthus powellii*) and redroot pigweed (*Amaranthus retroflexus*) in Ontario show different patterns of resistance to ALS inhibiting herbicides and to triazines. *Weed Sci. Soc. Am. Abstr.* 40:47-48.
- Ferguson, G. M., A. S. Hamill, and F. J. Tardif. 2000b. ALS inhibitor resistance in populations of *Amaranthus powellii* and *A. retroflexus*. *Weed Sci.* (in press).
- Friesen, L. F., T. L. Jones, R. C. Van Acker, and I. N. Morrison. 2000. Identification of *Avena fatua* populations resistant to imazamethabenz, flamprop, and fenoxaprop-P. *Weed Sci.* 48:532-540.
- Friesen, S. and J. C. Hall. 2000. Confirmation of resistance to fenoxaprop-ethyl, imazamethabenz-methyl, and flamprop-methyl by two wild oat populations. *Weed Sci. Soc. Am. Abstr.* 40:61.
- Goodwin, M. 1994. An extension program for ACCase inhibitor resistance in Manitoba: a case study. *Phytoprotection* 75(Suppl.):97-102.
- Hall, L. M., K. M. Stromme, G. P. Horsman, and M. D. Devine. 1998. Resistance to acetolactate synthase inhibitors and quinclorac in a biotype of false cleavers (*Galium spurium*). *Weed Sci.* 46:390-396.
- Hall, J. C., J. T. O'Donovan, and L. M. Hall. 1999. Basis for resistance of a wild mustard (*Sinapis arvensis* L.) biotype to ethametsulfuron-methyl. *Weed Sci. Soc. Am. Abstr.* 39:123.
- Heap, I. M. 2001. International Survey of Herbicide-Resistant Weeds. [Online] <http://www.weedscience.com> [accessed February 10, 2001].
- Heap, I. M. and I. N. Morrison. 1992. Resistance to auxin-type herbicides in wild mustard (*Sinapis arvensis* L.) populations in western Canada. *Weed Sci. Soc. Am. Abstr.* 32:55.
- Heap, I. M. and I. N. Morrison. 1996. Resistance to aryloxyphenoxypropionate and cyclohexanedione herbicides in green foxtail (*Setaria viridis*). *Weed Sci.* 44:25-30.
- Jasieniuk, M., I. N. Morrison, and A. L. Brulé-Babel. 1995. Inheritance of dicamba resistance in wild mustard (*Brassica kaber*). *Weed Sci.* 43:192-195.
- Jeffers, G. M., J. T. O'Donovan, and L. M. Hall. 1996. Wild mustard (*Brassica kaber*) resistance to Ethametsulfuron but not to other herbicides. *Weed Technol.* 10:847-850.
- 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.
- 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. *Abstr. Third International Weed Science Congress, Foz do Iguassu, Brazil.* Corvallis, OR: International Weed Science Society. p. 144.

- Morrison, I. N., L. Bourgeois, L. Friesen, and D. Kelner. 1995. Betting against the odds: The problem of herbicide resistance. *In* T. L. Roberts, ed. Proc. 1995 Western Canada Agronomy Workshop, Red Deer, AB: Potash and Phosphate Institute of Canada. pp. 159-164.
- Morrison, I. N. and M. D. Devine. 1994. Herbicide resistance in the Canadian prairie provinces: five years after the fact. *Phytoprotection* 75(Suppl.):5-16.
- O'Donovan, J. T., M. N. Baig, and J. C. Newman. 2000. Persistence of herbicide resistance in wild oat (*Avena fatua* L.) populations after cessation of triallate or difenzoquat use. *Can. J. Plant Sci.* 80:451-453.
- O'Donovan, J. T., G. M. Jeffers, D. Maurice, and M. P. Sharma. 1994a. Investigation of a chlorsulfuron-resistant chickweed [*Stellaria media* (L.) Vill.] population. *Can. J. Plant Sci.* 74:693-697.
- 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.
- O'Donovan, J. T., M. P. Sharma, K. N. Harker, D. Maurice, M. N. Baig, and R. E. Blackshaw. 1994b. Wild oat (*Avena fatua*) populations resistant to triallate are also resistant to difenzoquat. *Weed Sci.* 42:195-199.
- Saint-Louis, S., A. DiTommaso, and A. K. Watson. 2000. Resistance of common ragweed (*Ambrosia artemisiifolia* L.) to the herbicide linuron in carrot fields in southwestern Québec. *Weed Sci. Soc. Am. Abstr.* 40:92.
- Shirtliffe, S. J., M. H. Entz, and B. D. Maxwell. 1998. The effect of chaff collection and harvest timing on the seed spread of wild oat (*Avena fatua*). *Weed Sci. Soc. Am. Abstr.* 38:39.
- Smisek, A., C. Doucer, M. Jones, and S. Weaver. 1998. Paraquat resistance in horseweed (*Conyza canadensis*) and Virginia pepperweed (*Lepidium virginicum*) from Essex County, Ontario. *Weed Sci.* 46:200-204.
- Switzer, C. M. 1957. The existence of 2,4-D resistant strains of wild carrot. *Proc. North Eastern Weed Control Conf.* 11:315-318.
- Van Eerd, L. L., G. R. Stephenson, and J. C. Hall. 2000. 2,4-D resistance in wild carrot (*Daucus carota*). *Weed Sci. Soc. Am. Abstr.* 40:64.
- Veldhuis, L. J., L. M. Hall, J. T. O'Donovan, W. Dyer, and J. C. Hall. 2000. Metabolism-based resistance in a wild mustard (*Sinapis arvensis* L.) biotype to ethametsulfuron-methyl. *J. Agric. Food Chem.* 48:2986-2990.