

How did we get here? Principles of Herbicide Resistance in Weeds

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Human Nature

Humans have evolved because of their inclination to repeat successful practices. If a wheat variety produces a high yield, then we are likely to grow the same variety again. If a herbicide controls weeds, we are inclined to use the same product again. However, pests, including weeds, adapt to repeated practices. Pests adapt or are selected to fill the niche our cropping practices create. We then need to alter our cropping practices to close the niche. This is the origin of the game we have been playing with weeds since the beginning of cropping agriculture.

Resistance Nature

Within a species that is largely susceptible to (controlled by) a herbicide, a few individual plants may have randomly altered genes that confer resistance to the herbicide. The genetic basis for resistance means that weeds resistant to a herbicide can pass these traits on to future generations. To be termed resistant, the weed species must formerly be controlled by the herbicide. This definition eliminates weeds that are consistently tolerant to a herbicide, or weeds that survive a herbicide because of environmental or staging factors.

Herbicide Resistance: inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the susceptible type

Herbicide Nature

Not all herbicides are alike in their ability to select for herbicide resistant weeds. Some, like Group 4 herbicides 2,4-D and MCPA, have been used successfully for over 50 years with limited selection of resistant weeds. Others, like the Group 2 herbicides Glean (chlorsulfuron) and Ally (metsulfuron methyl), were on the market only a few years before there were substantiated reports of resistance.

Several factors are responsible for this difference in risk associated with herbicide groups. Herbicides vary in their effectiveness. Effective herbicides place more selective pressure on weed populations than do less effective herbicides. Similarly, residual herbicides place a longer duration of selection pressure on weeds than do non-residual ones. The longer a herbicide is effective, the more the field population is depleted of susceptible plants, leaving only resistant ones to increase.

However, these factors do not explain all of the differences between herbicides. To provide further explanation we must describe the way in which weeds can be selected to be resistant to a herbicide.

Metabolism based resistance – Plants have the ability to degrade some herbicides before they cause harm. This is the basis of selectivity between weed and crop of many herbicides. Selective herbicides can enter crop plants, but are rapidly broken down or shunted away and do not control the crops. When the same herbicide enters a weed, it is not degraded quickly and the weed is controlled. For example, wheat metabolizes Group 1 (grass herbicide) products like Horizon (clodinofof) but wild oat does not metabolize the herbicide and is controlled. Repeated applications of Horizon can screen the wild oat population for any individual plants that have the ability to metabolize the herbicides. These rare individuals survive and reproduce, increasing in number. Herbicides such as the Group 1 products which are readily degraded are more likely to select for weeds with metabolism-based resistance. Conversely, it is highly unlikely that weeds will be selected to metabolize non-selective herbicides like glyphosate.

Site of action based resistance – Herbicides generally need to bind to a site of action, usually an enzyme, to have an effect on a weed. Weeds can become resistant if the site has been altered by a chance mutation so the herbicide cannot bind. Some enzyme sites probably have more ability to withstand modification than others without interrupting a critical process to cause plant death. For example, the Group 2 herbicides like Ally and Pursuit inhibit an enzyme called ALS which can be modified in many ways without causing plant death. These target sites are thus more likely to confer resistance than less-modifiable binding sites for herbicides like 2,4-D or glyphosate. Researchers speculate that either 2, 4-D binds to many different sites in the plant and/or that site modification is either lethal or causes the plant to be unfit. Very few changes to the site of action of glyphosate confer resistance by excluding the glyphosate molecule, and most of those decrease the ability of the resistant plant to survive.

Alternative mechanism of resistance - Other mechanisms of resistance occur but they are less common than metabolism or changes to the site of action. These include changes in uptake or translocation, or sequestration of the herbicide away from the site of action. It is speculated that one of these alternative methods is responsible for glyphosate resistance in Australian rye grass populations.

Genetic nature of the resistance mechanism – The nature of herbicide resistance determines the initial frequency of resistance genes in weed populations, for example; one in a million or one in a billion. This starting point determines the time required for selection of resistance to reach a noticeable size in a field. Rare resistance takes longer to form a significant part of the population than more frequent resistance. It also determines how many fields will randomly contain the resistant weeds. We know that resistance to Group 1 and 2 herbicides (grass herbicides and ALS inhibitors, respectively) can be repeatedly selected, even within the same field. We expect that glyphosate resistance will be more rare.

Secondarily, either dominant or resistant genes can confer resistance, but dominant are more common. Dominant genes also tend to increase more quickly in the population because both homozygous (RR) and heterozygous (Rr) gene pairs make the individual resistant, and any crosses with a dominant gene (R) will produce resistant progeny.

Fitness of resistant plants

In many instances, there is little difference in the ability of the resistant and susceptible individuals to survive and produce seed. However, for some types of resistance such as atrazine, some resistant weeds are less fit and less capable of reproducing than the susceptibles. The implications of reduced fitness are that in the absence of selection pressure (the herbicide),

there may be a decline in the resistance frequency of the weed population. This is uncommon on the prairies, and therefore we rarely see a decrease in frequency of resistant types in the population whether there is subsequent herbicide selection or not.

Weed Nature

Weeds vary in their density in fields. In central Alberta, chickweed is very abundant while in Manitoba, green foxtail is found in large numbers (Leeson et al 2002). Weeds found in large numbers are more likely to have a resistant individual that can be selected. It is a numbers game, let's call it Russian thistle roulette.

This general rule is modified by the genetic diversity of the weeds. Some weeds, especially those which are outcrossing rather than self fertilizing, are more variable than others. They are more likely to contain an individual with a resistant mutation. In contrast, those species with a long seed bank life are somewhat slower to be selected for resistance. Each year any new resistant individuals remain at a low frequency compared to the large numbers of susceptible plants that emerge from the seed bank, and buffer the increase of resistance.

Relative Risk of Resistance

The risk of resistance varies both with herbicide and with weeds. This is summarized in Table 1. Consider the risks when choosing products, and select low risk products where possible. Use high risk products sparingly, especially when high risk weeds require control.

Table 1. Relative risk of selection of herbicide resistance by herbicide group and weed species.

	Wild Oat	Green foxtail	Wild mustard	Cleavers	Spiny annual sowthistle	Chickweed	Red root pigweed	Kochia	R	th
Group 1	High	High	n/a	n/a	n/a	n/a	n/a	n/a	n	n
Group 2	High	High	High	High	High	High	High	High	H	H
Group 3	Low	Moderate	n/a	n/a	n/a	Low	Low	Low	L	L
Group 4	n/a	n/a	Moderate	Low	Low	Low	Low	Low	L	L
Group 5	n/a	n/a	Moderate	Moderate	Moderate	Moderate	Moderate	Moderate	M	M
Group 6/7	Low	Low	Low	Low	Low	Low	Low	Low	L	L
Group 8	Moderate	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n	n
Group 9	Low	Low	Low	Low	Low	Low	Low	Low	L	L
Group 10	Low	Low	Low	Low	Low	Low	Low	Low	L	L
Group 22	Low	Low	Low	Low	Low	Low	Low	Low	L	L

n/a – Products do not usually control these weeds and therefore do not select for resistance

Current State of Herbicide Resistant Weeds

The number of resistant weeds and the mechanisms of resistance are determined in Canada by laboratory studies of herbicide resistance mechanisms, by compiling the results of testing facilities and through resistant weed surveys. While no single method provides a comprehensive picture, put together we have a blurry image emerging (Table 2).

Many broadleaf weeds have been selected for group 2 resistance. The location of resistant populations reflects the areas in which they are abundant. Of more concern are Group 1 resistant wild oat and green foxtail (Figures 1 and 2). These weeds are of concern because of the few alternative herbicides available for their control. Group 1 resistant wild oat are found commonly in the black soil zone, where wild oat are abundant and Group 1 product use has been high. To date, Group 1 resistant green foxtail is located primarily in Manitoba, but this trend is unlikely to continue.

Table 2. Herbicide resistant weeds found in the Prairie Provinces

Herbicide Group	Resistant Biotype	Mechanism
Group 1	Wild oat	Target site/ Metabolism/Both
	Green foxtail	Target site
Group 2 [stinkweed –target site; redroot pigweed-unknown; green foxtail-unknown]	Wild mustard	Target site/ Metabolism
	Wild oat	Unknown
	Ball mustard	Unknown
	Chickweed	Target site
	Kochia	Target site
	Russian thistle	Target site
	Spiny annual sowthistle	Target site
	Cleavers	Target site
	Hempnettle	Unknown
Group 3	Green foxtail	Site of action
Group 4 [hemp-nettle]	Wild mustard	Unknown
Group 5	Wild mustard	Site of action
Group 8	Wild oat	Unknown
Multiple Groups (1, 2, 8, 25)	Wild oats	Metabolism
Multiple Groups (2, 4?)	Cleavers	Site of action and unknown

Figure 1. Group 1 (APP and CHD) resistant wild oat as determined by samples sent to resistance testing facilities. Legend refers to the number of fields with resistant plants (Beckie et al 2003).

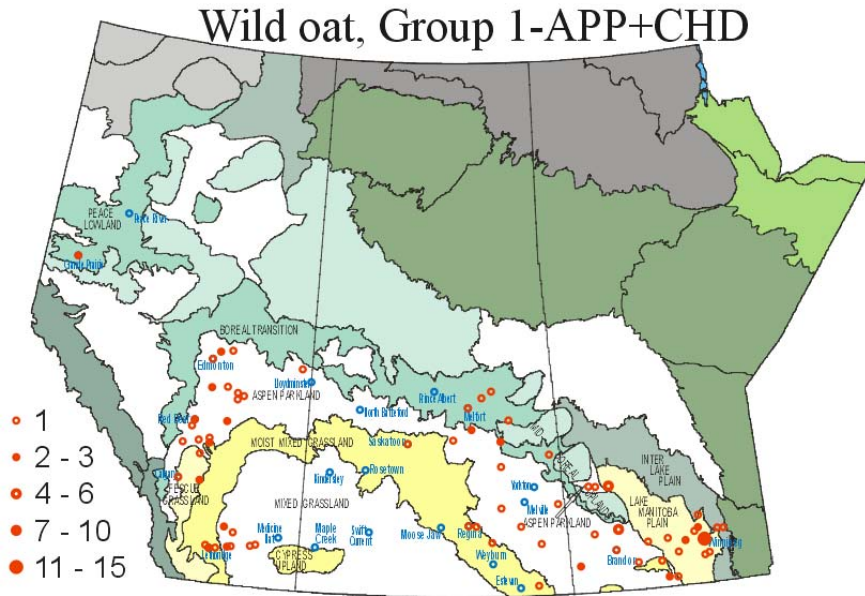
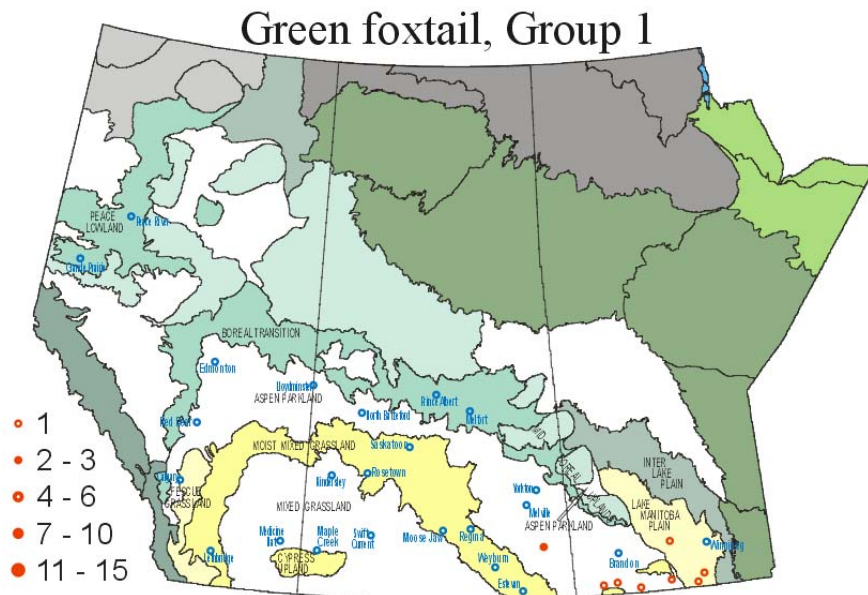


Figure 2. Occurrence and location of Group 1 resistant green foxtail in the Prairie Provinces (Beckie et al 2004).



Conclusion

By understanding the principles of selection of herbicide resistance, growers can sort through the many options they have to reduce the risk of resistance on their farm. However, for every 'rule' there is an exception. The diversity of strategies that weeds employ can only be countered by diversifying our cropping practices.

Other Information on Principles of Herbicide Resistance

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