BATTLE RIVER IMPLEMENTS LTD.

JANUARY AGRONOMY UPDATE

HERBICIDE RESISTANCE

Credit: Canola Watch

If it sometimes seems like the list of weeds resistant to some of our most widely used herbicides is growing longer every year, that's because it is. The arsenal of active ingredients available to us has not changed much since the 1980s. In the meantime, constant selection pressure on the weed populations by the use of these herbicides has had an inevitable result. According to Dr. Charles M. Geddes, a research scientist with Ag Canada working out of the Lethbridge Research and Development Centre, an average of 1.7 new weed bio-types are identified as resistant to at least one herbicide each and every year in Canada.

As a result, we now have about 80 or so weeds in Canada alone that have developed resistance to herbicides. On the Prairies, that list includes documented resistance to Groups 1,2,3,4,5,9,14 and 15; in other words, just about everything we use. We have been dealing with herbicide resistance for about 50 years now, and I can say with confidence that no silver bullet is coming to save us. Managing resistance comes down to herbicide rotation, layering of effective rates of multiple active ingredients and using Integrated Pest Management practices that can help our crops out-compete the weeds. The basis of understanding how to manage any problem is to understand the ins and outs of the issue, so I think it's important to review how resistance develops in a population, because that will influence our management practices.

The most common form of resistance we see on the Prairies is called *Target Site Mutation.* Our herbicides generally work by interfering with the biological processes of the weeds; depriving them of the means of obtaining the resources they need to survive and grow. This often means interfering with the enzymes that control the plant's biological functions. Herbicides work by blocking the sites where enzymes would normally be doing their job, causing the weeds to die. Target Site Mutation is what happens when the plant alters those sites so that the herbicide can no longer block the enzymes.Group 1 ACCase resistance is an example of this. The plant can also get around the herbicide by duplicating the number of target sites the chemical has to cover. It replicates enough sites that the herbicide can't cover them all without a massive increase in the application rate. This is what is happening in kochia when it develops resistance to glyphosate.

The second kind of resistance that can develop is called *"Non Target-Site Resistance".* This is when the plant does things that interfere with the herbicide uptake and movement, preventing it from ever reaching the target sites. This can take a few different forms. The plants can actually be selected over time to alter leaf angles or structure or select for hairier leaves – anything that increases the chances of the herbicide running off the surface and decreasing the absorption of the chemical. Or it can intercept the herbicide inside the plant before it reaches the target site through something called vacuolar sequestration, which sounds complicated but really isn't. Each cell in every plant has a storage area called a vacuole. Think of it as the plant's answer to that spare closet in your home where you throw all the junk you don't know what to do with, but just can't bring yourself to throw away. The plant recognizes the herbicide as a foreign invader and shunts it off to the vacuole where it can be stored in isolation from the plants' biological processes, rendering it harmless. Some weeds can also selectively reduce the amount of chemical trans locating throughout the plant. Herbicides usually spread throughout the plant via the water and nutrient distribution systems. Some weeds show the ability to isolate the area where the herbicide has entered the plant and physically prevent it from spreading by stopping all water and nutrients in or out of that area – the plant's equivalent of doing an amputation to save its life. Kochia has shown the ability to do this with Dicamba based herbicides.

The final, and possibly most concerning pathway to resistance is through increased metabolism. Using a reduced rate of herbicide can put pressure on a weed population that selects for plants that are able to metabolize the herbicide into less toxic metabolites quickly enough that the plant survives the treatment. While this tends to be a slow selection process, once a population is established, it has the potential for high levels of cross mutation, making herbicide rotation less effective. It has even been theorized that plants with this type of metabolic resistance could be successful in metabolizing herbicides from groups they have never been exposed to previously at all.

This last type of resistance isn't widely understood yet, but it is the reason that researchers like to emphasize layering of **effective rates** of multiple active ingredients. So keep eyes out for those prepackaged products which reduce rates of some of the active ingredients as a cost saving measure. The long term price you pay may be more than you bargained for.