Canadian Agronomist
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Triallate-resistant wild oat also cross resistant to four other Groups

CATEGORY weeds | *December 12, 2018*

Herbicide screening studies on triallate-resistant (Group 8) wild oats found cross-resistance to Group 1 and Group 2 herbicides. Additionally, unexpected resistance to Group 14 and Group 15 herbicides was found even though the wild oats had never been previously exposed to these herbicides.

Two relatively new active ingredients have been registered for suppression or control of wild oats. Pyroxasulfone (Group 15) is a pre-emergent herbicide found in herbicides Focus, Fierce, and Authority Supreme. Sulfentrazone is a pre-emerge Group 14 herbicide found in Authority, and Authority Charge. These herbicides are valuable tools to help manage herbicide resistance.

However, because cross-resistance to triallate and pyroxasulfone was confirmed in annual ryegrass in Australia, research was conducted in western Canada to determine if wild oat populations that are resistant to triallate could also be cross-resistance to pyroxasulfone and/or sulfentrazone.

Group 1, Group 2 and Group 8 multiple resistance

Two wild oat populations with triallate resistance, an HR08-210 population from Olds, AB and an HR11-151 population from Rivers, MB, were tested for cross-resistance to Group 1 and Group 2 herbicides. Populations were screened with the Group 1 ACCase inhibitor herbicides fenoxaprop and quizalofop, and Group 2 ALS inhibitor herbicides imazamethabenz and imazapyr.

Fenoxaprop and imazamethabenz are selective herbicides and metabolized in wheat, while quizalofop and imazapyr are not metabolized. If a biotype is resistant only to the herbicide that is metabolized, it is an indication that enhanced metabolism may be responsible for resistance rather than a target-site mutation.

The wild oat populations were resistant to both Group 1 and Group 2 herbicides. The populations exhibited resistance to both Group 1 herbicides, indicating the presence of enhanced metabolism and target site mutation resistance mechanisms. Both populations were highly resistant to Group 2 imazamethabenz, but susceptible to imazapyr. This suggests that resistance to ALS inhibitors was only due to enhanced metabolism and not a target site mutation.

Unexpected cross-resistance to Group 14 and Group 15

The wild oat populations were then tested for resistance to pyroxasulfone and sulfentrazone. Despite the wild oat populations never having been exposed to pyroxasulfone or sulfentrazone, the HR11-151 wild oat population showed cross-resistance to both herbicides. However, the HR08-210 population was not classified as resistant to either pyroxasulfone or sulfentrazone.

Since the wild oat populations had never been exposed to pyroxasulfone or sulfentrazone the possibility of target-site mutations conferring multiple-resistance mechanisms for these herbicides is unlikely, as they have different target sites. Cross-resistance is believed to be a result of resistance mechanisms that had previously been selected for by ACCase and ALS inhibitors (enhanced metabolism) or triallate (enhanced endogenous gibberellins).

Enhanced metabolism would allow the wild oat to breakdown the herbicide before it becomes lethal to the plant, while increased endogenous gibberellin levels may increase the rates of germination and seedling growth, resulting in the avoidance of toxicity across these soil-applied herbicides.

Since only one of two wild oat populations exhibited resistance to pyroxasulfone and sulfentrazone, further research is required to determine if enhanced metabolism and endogenous gibberellins is a common occurrence in wild oat cross-resistance. In the meantime, agronomists and farmers need

to be aware of the potential for cross-resistance between current Group 1, 2 and/or 8 resistant wild oat field populations and Groups 14 and 15 herbicides when planning herbicide rotations.

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Mangin, A.R., Hall, L.M., Beckie, H.J. 2017. Triallate-resistant wild oat (*Avena fatua* L.): unexpected resistance to pyroxasulfone and sulfentrazone. Can. J. Plant Sci. 97: 20-25. https://doi.org/10.1139/cjps-2016-0029