



IRRIGATION RESEARCH & EXTENSION COMMITTEE

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Herbicide resistance mechanisms and common HR misconceptions

ANDREW STORRIE

Weeds Agronomist
NSW Department of Primary Industries
Tamworth Agricultural Institute
Calala NSW

Ph: 02 6763 1174

Email: andrew.storrie@dpi.nsw.gov.au

IREC

C/- CSIRO Land and Water, Griffith
Private mail bag 3 Griffith NSW 2680

Tel: 02 69601550 **Fax:** 02 69601562 **Email:** irec@irec.org.au

Take home message

Surveys have shown many growers and agronomists still don't understand the basic mechanisms behind herbicide resistance and still confuse poor herbicide control with resistance.

Successful long term weed management relies on the integration of a range of techniques to keep weed numbers low. The key additional factors that growers need to implement are monitoring levels of control and preventing survivors from setting seed.

The genetic basis for resistance is usually governed by major single genes (target-site resistance), however glasshouse research has shown that repeated use of low herbicide rates can lead to resistance, most likely controlled by the additive effects of multiple minor genes (non-target-site resistance).

Why worry?

Not understanding the basic mechanisms of resistance will make weed management very difficult because incorrect decisions will be made. History has shown that the common way to manage resistance is to go to a new mode-of-action (MOA) group. History also shows that once you have resistance to multiple groups (multiple resistance), farming options become limited and more expensive.

Mechanisms of resistance

Types of resistance are normally divided into target site and non-target site resistance.

Target site resistance – is the dominant form of resistance and is caused by a mutation on the enzyme (a change in the shape) at the site where the specific herbicide binds, thus stopping the plant's normal biochemical processes. For example, Group B herbicides bind to the substrate binding site for the acetolactate synthase (ALS) enzyme, thus preventing the production of essential amino acids. Without these amino acids, the plant eventually dies.

This type of resistance is controlled by major genes and tends to be like a "light switch". Resistant plants are usually unaffected by commercial rates of herbicide and susceptible individuals die quickly. Target-site resistant and susceptible plants are equally "competitive".

Non-target site resistance – is also referred to as metabolic resistance and tends to include a number of mechanisms other than target-site resistance. These mechanisms include reduced herbicide absorption, reduced translocation within the plant, reduced herbicide activation, enhanced herbicide detoxification (P450 enzymes), changes in intra- or intercellular compartmentalisation, and enhanced repair of herbicide-induced damage.

This type of resistance is generally controlled by a number of minor genes.

Neve & Powles (2005) demonstrated the possible rapid development of non target-site resistance by taking a known susceptible population of annual ryegrass and treating it with a one tenth rate of diclofop. The survivors were cross pollinated. This process was continued through another two generations. There were sufficient seedlings in the fourth generation to be classed resistant when treated with the full rate of diclofop. This was a result of concentrating (stacking) a number of minor genes conferring resistance through cross pollination.

So will this happen in commercial paddocks? It is unlikely for at least two reasons. Firstly most farmers use robust rates of herbicide that will kill these "slightly" resistant plants. Secondly, the gene pool will be "diluted" by untreated ARG plants that emerge after the early post emergent herbicide has been applied, and a percentage will not emerge until the following year.

Multiple resistance – is a term that describes weed populations with resistance to more than one herbicide mode-of-action (MOA) groups. There is a population of annual ryegrass that exhibits resistance to five herbicide MOA groups and a population of wild radish resistant to four MOA groups.

Misconceptions

In 2005 growers and agronomists in the Northern Grain Region were surveyed as part of the Northern Herbicide Resistance Project DAQ 527. The aim of the survey was to find out what farmers and agronomists had learned over the duration of the project and where there were still gaps in knowledge or misunderstandings regarding herbicide resistance.

Poor control or resistance? – Many in the industry are still confused by the difference between poor control and herbicide resistance, particularly with glyphosate. Examples of poor control being confused with glyphosate resistance included the species black bindweed, nutgrass, sowthistle, annual ryegrass and fleabane.

Many growers are trying to control large weeds, often when temperatures are high and low relative humidity. Plants are stressed, with thickened cuticles, and up to 50% of the herbicide is not reaching the target due to evaporation, so coverage and actual dose rate are greatly reduced.

Herbicide MOA Groups – Northern NSW farmers and agronomists have a better understanding of MOA groups than their Queensland counterparts, probably due to the higher levels of resistance already present, however as shown in Table 1, only 28% of those surveyed could name a Group C herbicide and 21% a Group L herbicide.

Table 1. Percentage of respondents able to name herbicides with each MOA group.

MOA group	SQ	NNSW
M	10	72
A	20	66
B	13	72
C	13	28
L	10	21
I	13	48

Practices that increase the risk of HR – Given two situations to choose from and asked to nominate which one increased the risk of resistance, respondents gave mixed responses and revealed that their knowledge of such issues is lacking.

The use of herbicides tank-mixes were not well understood, as was the risk of treating high weed densities with herbicides. Agronomists had a better understanding than farmers.

Respondents understood that they should not rely on one MOA group however did not understand that relying on rotating herbicide groups would only delay HR.

Reversion of herbicide resistance – nearly 40% of respondents felt that HR would only last for five years if herbicides were not used with the weed populations reverting to susceptible.

Where does resistance come from? – While the spraying regime of the grower is the greatest cause of herbicide resistance, growers themselves did not rate this avenue of resistance very highly. Agronomists fared much better. This finding suggests that growers continue to believe that resistance comes from external elements, largely beyond their control.

The “Talisman” Approach to herbicide resistance prevention

Experience over the past 10 years has shown that many within agriculture are human, and rely on a single practice as a “Talisman” to protect them from herbicide resistance. History however shows that, despite this type of approach being popular, it DOES NOT work.

We know for example that growing summer crops is a sure way of preventing the development of HR. Experience shows that simply growing summer crops without monitoring levels of weed control and preventing “escapes” from setting seed will lead to the development of resistant weeds, particularly in a “no-till” system.

Therefore cultivation must be the answer. Well, no. The cultivation must be targeted at reducing a significant proportion of the problem species in question. Cultivating in summer will not solve winter weed problems and vice versa. Cultivation could be used to “stimulate” the germination of weeds. Cultivating a flush of weeds prior to sowing will only help manage resistance if the weeds actually die.

No one technique will solve the herbicide resistance issue.

Growers must:

- 1. Keep weed numbers low***
- 2. Monitor levels of control***
- 3. Stop survivors setting seed***