

## Section 2: Herbicide resistance

**Herbicide resistant weed populations are now found throughout all cropping areas of Australia from Western Australia to central Queensland. Currently, 25 weed species in Australia have been identified as being resistant to one or more herbicide mode-of-action (MOA) groups.**

**The number of resistant species and areas affected by resistance will continue to increase until integrated weed management (IWM) practices are widely adopted in Australian cropping systems.**

### Herbicides

The first herbicide was released onto the Australian market in 1946. Cost-effective and simple, herbicides quickly became the most heavily relied upon weed control method for many farmers. Even today, in some instances, using herbicides can represent the only conscious weed management decision made by farmers.

Along with an associated decline in use of alternative weed control methods (such as cultivation), heavy herbicide use has resulted in high selection pressure for herbicide resistance in populations of weed species.

By understanding the implications and evolutionary processes of herbicide resistance, appropriate weed management strategies can be devised that will minimise the impact of herbicide resistant weeds and delay development of further resistance.

This section of the manual deals with herbicide resistance that has developed in weeds through over-reliance on herbicide control. For information on herbicide tolerant crops see *Section 3: Agronomy 3 Herbicide tolerant crops*.

### What is herbicide resistance?

It is important to differentiate between herbicide resistance and herbicide tolerance.

Herbicide *resistance* is the inherited ability of a *plant* to survive and reproduce following exposure to a dose of a herbicide normally lethal to the wild type (Heap 2006).

Herbicide resistance does *not* equate to poor performance of a herbicide. Resistant weeds can often survive application of herbicide at rates that are much greater than the recommended rate.

Herbicide *tolerance* is the inherent ability of a *species* to survive and reproduce after herbicide treatment at a normal use rate (Heap 2006). There is no selection involved (through herbicide application) because the species is naturally tolerant.

### Herbicide resistance fact box

- Resistance is the inherited ability of an individual plant to survive a herbicide application that would kill a normal population of the same species.
- Twenty five weed species in Australia currently have populations that are resistant to at least one herbicide mode-of-action (MOA) group.
- Seven distinct MOA groups are no longer effective against one or more resistant weed populations.
- Herbicide resistance is normally present at very low frequencies in weed populations before the herbicide is first applied. Variation exists within every population, with some individuals having the ability to survive the herbicide application.
- The frequency of resistant individuals within a population will vary greatly within and between species and MOA groups.
- A weed population is defined as resistant when a herbicide that once controlled the population is no longer effective (sometimes an arbitrary figure of 20% survival is used).
- The proportion of herbicide resistant individuals will rise due to selection pressure in situations where one herbicide MOA group is applied repeatedly.
- Herbicide resistance is *permanent* in weeds and their progeny with dominant target-site resistance. With cessation of the use of that herbicide MOA group, the ratio of dominant target-site resistant to susceptible individuals will remain the same – only the total number of weeds present can be reduced. Weeds with this type of resistance do not exhibit a fitness penalty.

Examples of herbicide tolerance include wheat or *Vulpia* spp. to post-emergent applications of diclofop.

Herbicide tolerance is the underlying feature of herbicide selectivity. It enables crops to survive applications of selective herbicides and explains why selective herbicides do not kill all weeds.

## Commonly used terms

### Herbicide mode-of-action groups

Herbicides act by targeting specific plant processes. This process-specific activity is termed 'mode of action' (MOA). In Australia all herbicides are classified into groups based on their MOA and named with a group letter from A to N. MOA group classifications can be found on all herbicide labels, to identify the group to which a herbicide belongs.

MOA groups are ranked according to the risk of weed populations becoming resistant to those herbicides. Groups A and B are high risk while the remainder are moderate risk.

### MOA subgroup chemical classes

Within a herbicide MOA group there may be two or more subgroups. Subgroups are usually based on the different chemical classes that inhibit the same enzyme. Group Z contains those herbicides that do not fit elsewhere.

### Selection pressure

Selection pressure is a term used to describe the amount of selection for resistance applied by the herbicide application. Every time herbicide is used, susceptible individuals are killed and resistant individuals survive. The greater the number of susceptible individuals killed by the herbicide, the higher the selection pressure.

### Resistance mechanisms

This term is used to describe the specific processes that enable the plant to survive an application of herbicide. Resistant populations of weeds may have either target-site or non-target-site resistance.

### Target-site resistance

Target-site resistance occurs when there is an alteration at the target-site. The alteration occurs at the normal herbicide site of action within the plant and is in the form of either a structural or biochemical change. This means that the herbicide will no longer be able to bind to its site of action, allowing the plant to survive the herbicide treatments.

### Non-target-site resistance

Non-target-site resistance (also referred to as *metabolic resistance*) is used to describe mechanisms other than changes at the target-site which enable an individual plant to survive a herbicide application. The potential mechanisms include reduced herbicide uptake, reduced translocation, reduced herbicide activation, enhanced herbicide detoxification, changes in intra- or inter-cellular compartmentalisation and enhanced repair of herbicide-induced damage.

### Cross-resistance

Cross-resistance is defined as the ability of a weed population to express resistance to more than one herbicide. It may arise without the weed population ever being exposed to one of the herbicides. There are two types of cross-resistance:

1. *Across herbicide subgroups*. This occurs when a weed population is resistant to more than one herbicide subgroup within a specific MOA group. For example, populations of wild oats that are resistant to Group A 'fops' may also be resistant to Group A 'dims', even though they have not been exposed to a herbicide from the 'dim' subgroup. This is usually *target-site* resistance.
2. *Across herbicide mode-of-action groups*. This occurs when a weed population is resistant to herbicides from within more than one MOA group. For example, a population of annual ryegrass selected only by Group A herbicides may become resistant to both Group A and Group B herbicides. This is usually *non-target-site* resistance.

### Multiple resistance

Multiple resistance is a term used to describe weed populations that exhibit more than one resistance mechanism, allowing the plant to withstand herbicides from different subgroups. Some populations of resistant annual ryegrass possess both target- and non-target-site resistance. For example, one population of annual ryegrass present in Australia exhibits confirmed resistance to five different herbicide MOA groups.

### Partial resistance or developing resistance

Partial resistance and developing resistance are terms used to describe a situation where only a small proportion (often less than 20%) of the population survives the standard application rate of the herbicide in question. Weed populations are normally classified by testing services as resistant when more than 20% of the population survives the standard application rate of herbicide.

## How does a weed become resistant to a herbicide?

There are three major ways in which resistance may arise within a weed population:

1. *Pre-existing resistance.* Within any weed population there may be some plants that already contain a rare change in a gene (or genes) that enable them to survive the application of a particular herbicide that would normally kill this species.

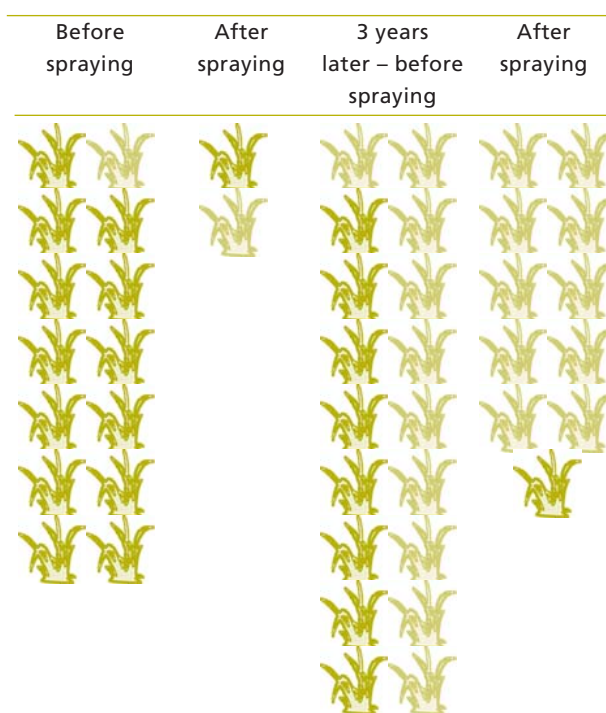
Genetic variation may alter physiological traits that enable herbicide uptake, translocation and activation at the site of action. Alternatively, changes may influence the plant's ability to detoxify herbicides, or enable transport to a site within the plant where the herbicide is not lethal.

Each time the herbicide is applied, susceptible plants die and those with resistance survive (Figure HR1).

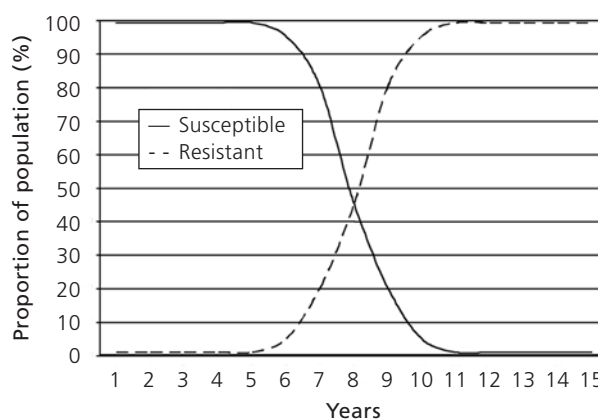
The initial frequency of plants with pre-existing resistance is usually very low. Therefore, the majority of plants in a wild weed population will be susceptible to herbicides effective on that species. Persistent use of herbicides with the same MOA will kill the susceptible portion of the population, resulting in the gradual increase in the proportion of resistant individuals (Figure HR2).

This process is described as applying selection pressure. By removing susceptible plants from the population, plants that can survive application of the herbicide at the given rate are selected.

2. *Importation of resistance.* It is possible that resistance may not be present in the population initially, but is introduced as a weed contaminant in crop seed or fodder, on machinery or on/in animals. This is particularly important for 'rarer' forms of resistance such as glyphosate resistance.
3. *Natural dispersal.* Weed seeds can also be spread by wind and water; for example, species such as sowthistle and fleabane can be spread up to 20 m by the wind. Pollen can also be dispersed great distances although the percentage remaining viable at distances greater than 10 m is low. Floodwater also has the potential to move a wide range of weed seeds over large distances. Dispersal by these mechanisms can increase the speed of development of herbicide resistance if integrated weed management (IWM) is not implemented.



**Figure HR1** Rare genes for herbicide resistance may pre-exist in a weed population. The proportion of resistant to susceptible weeds will change under selection pressure



**Figure HR2** The impact of repeated selection pressure (repeated application of herbicides with the same MOA) on herbicide resistance status (the proportion of plants susceptible or resistant to the herbicide)

## Factors influencing the development of resistance

Herbicide resistance is normally present in some individual plants of weed populations before herbicides are first applied. Several factors may affect the number of seasons in which a herbicide is applied before the general population becomes resistant. These are:

- initial frequency of resistance gene(s)
- MOA group of the herbicide being applied
- herbicide efficacy
- the size of the weed population

- the proportion of the weed population treated
- weed biological factors.

### Initial frequency of resistance gene

The frequency of individuals able to survive a herbicide application varies for different herbicides.

For example, high initial resistance in three untreated annual ryegrass populations (Table HR1) explains the rapid evolution of resistance to Group B herbicides in this weed species once the herbicides are used. This is due to the high numbers of individual plants able to survive and reproduce after herbicide application.

**Table HR1** Initial frequency of individuals resistant to two Group B herbicides in three previously untreated annual ryegrass populations (Preston and Powles 2002)

Herbicide MOA group	Active ingredient	Frequency range
Group B	sulfometuron-methyl	1 plant in 45,000 to 1 in 8,000
Group B	imazapyr	1 plant in 100,000 to 1 in 17,000

For other herbicides the initial frequency may be as high as 1 plant in every 10,000 or as low as 1 plant in every billion (Table HR2). Where initial frequencies of resistance are higher, fewer herbicide applications are necessary for resistance to develop.

**Table HR2** Initial frequency of resistant individuals of annual ryegrass, estimated by modelling (Diggle pers. comm. 2006)

	Estimated initial frequency
Group A eg diclofop-methyl	1 plant in 1,000,000
Group B eg chlorsulfuron	1 plant in 10,000
Group M eg glyphosate	1 plant in 1,000,000,000

Neve et al (2003) simulated the evolution of glyphosate resistance in annual ryegrass. Using an initial resistance frequency of 1 plant in one million, the model predicted resistance would evolve in all populations where glyphosate is used in less than 10 years. Changing the model parameters to make the resistance gene less frequent increased the length of time in which glyphosate would be effective to more than 10 years before resistance evolved.

### MOA group of the herbicide being applied

The number of times a herbicide can be applied prior to a weed population developing resistance will vary.

Some rules of thumb regarding the number of years of effective herbicide application before resistance evolves, according to MOA group of the herbicide being used, are given in Table HR3 (see also Table HR13).

In the case of herbicides such as triazines (Group C) and dinitroanilines (Group D), the frequency of individuals with a resistant gene enabling plants to survive the herbicide application is lower than for Group A and B herbicides. A longer period of exposure to the selection pressure (10 or more years of application) is required for weed populations to become resistant to these herbicides.

**Table HR3** Number of years of herbicide application before resistance evolves (based on Preston et al 1999)

Herbicide group	Years of application	Herbicide resistance risk
A	6–8	High
B	4	High
C	10–15	Medium
D	10–15	Medium
F	10	Medium
I	Not known	Medium
L	>15	Medium
M	15	Medium

The following Australian examples give an indication of the variation in time lag from initial herbicide application to development of resistance:

- Populations of annual ryegrass have developed resistance after only six applications of 'fops' (Group A) and four applications of sulfonylureas ('SU' – Group B) in Western Australia (Gill 1995).
- In New South Wales annual ryegrass has evolved resistance to glyphosate (Group M) after 15 years of application (Powles et al 1998) and elsewhere developed resistance to trifluralin (Group D) after 14 years of application (McAlister et al 1995).
- Wild oat populations have become resistant to 'fops' (Group A) and to a lesser extent 'dims' (Group A) after eight applications in most wheat growing areas of Australia (Mansoorji et al 1992) (Table HR4).
- Barley grass in South Australia has evolved resistance to paraquat (Group L) in no-tillage systems after approximately 15 years of application (Alizadeh et al 1998).
- Broadleaf weeds such as wild radish, Indian hedge mustard and common sowthistle have evolved resistance to 'SU' herbicides (Group B) after only 2–4 applications to weed populations in grain regions across Australia (Boutsalis and Powles 1995).

### Herbicide resistance case study #1

Group A cross-resistance to 'fops' and 'dims' in wild oats

*Weed:* A population of wild oats collected from the southeast of South Australia in 1989

*Rotation:* Cropped most years between 1981 and 1989 with a rotation primarily of wheat, canola and pasture legume seed crops

*Herbicide use history:* 1981–89 (Table HR4)

*Resistance profile:* Population cross-resistant to Group A herbicides, all 'fops' and some 'dims'

**Table HR4** Number of herbicide applications for South Australian herbicide resistance case study #1 (Mansooji et al 1992)

MOA group	Herbicide	Number of applications 1981–89
A (fop)	diclofop-methyl	3
	fluazifop-butyl	3
	haloxyfop-ethoxyethyl	2
D	trifluralin	3
J	triallate	1
M	glyphosate	1

In 1989 an application of haloxyfop (Group A) failed to control wild oats. Three weeks later, a second application of the same herbicide also failed.

In this case study heavy reliance on Group A ('fops') herbicide for annual grass control was typical practice. Tactic Group 2 (Kill weed(s) (seedlings) in the target area) was the primary focus for weed management, and failure to include tactics from alternative tactic groups led to development of herbicide resistance.

This study also shows that the overuse of a single MOA subgroup (in this case Group A 'fops') can lead to resistance in other MOA subgroups (in this case 'dims').

Triallate usually gives around 85% control of wild oats if applied correctly. The additional MOA herbicides listed in Table HR4 were applied to target other weed problems, rather than as a management technique for the Group A resistance. Trifluralin is not labelled for wild oat control (gives 60–70% control). The glyphosate was used as a pasture spray-top in one season.

### Herbicide efficacy

The level of kill or efficacy of the herbicide used will also affect resistance development. Highly efficacious herbicides exert strong resistance selection pressure. Modelling by Powles et al (1997) showed that herbicides resulting in 95% weed control increased the rate of resistance development to a greater extent than herbicides resulting in 80% weed control.

### The size of the weed population

The larger the number of weeds present, the greater the likelihood that there will be individuals with resistance to the herbicide within the population.

A useful analogy to understand the influence of size of weed population is the presence of white-flowered individuals in a Paterson's curse population. In a small population white-flowered individuals are unlikely to be present, but their numbers increase as population density increases. The gene controlling white flower colour is rare but, importantly, is already present in the population.

Similarly, genes controlling herbicide resistance are relatively rare. As with white-flowered Paterson's curse, the likelihood of resistant individuals being present will increase with increasing weed population. Unfortunately, unlike the white-flowered Paterson's curse, resistant plants look exactly the same as susceptible plants and will not be detected until they survive herbicide application.



Paddock of purple Paterson's curse with single white Paterson's curse flower circled. The white flower indicates a rare change in a gene. Photo: Steve Sutherland

### Herbicide rate and the development of resistance: does rate really matter?

Agronomists and growers often question whether high rates or low rates of herbicide lead to resistance.

Worrying about herbicide rate is irrelevant because both high and low rates can result in resistance if survivors are allowed to set seed. High rates can select for target-site resistance, and low rates tend to select for metabolic resistance.

When spraying with herbicides a high level of weed control should be targeted to avoid losses in crop yield. A high level of control is determined by herbicide efficacy rather than by rate. Whereas weed control in the order of 95% may be obtained under preferred spraying conditions, perhaps twice the recommended rate would be required to obtain the same level of control under poor spraying conditions.

It is important to use a robust rate for maximum weed kill, but it is also necessary to kill survivors using other tactics. Herbicides from the same MOA group should not be used frequently.

### The proportion of the weed population treated

If a greater proportion of the weed population is treated with the herbicide, more susceptible individuals will be killed and the selection pressure will increase. This might occur where multiple applications of the herbicide are made in one season, for example the use of glyphosate to control barnyard grass in northern summer fallows. It could also occur where a herbicide is applied late after more weeds have emerged, for example a late post-emergent application of metsulfuron to control broadleaf weeds in winter cereals. Herbicides with a long persistence in the soil such as chlorsulfuron (used as a pre-emergent herbicide on light-textured alkaline soils) can also increase the selection pressure.

### Weed biological factors

There are a number of key biological factors that will influence the number of years of herbicide application necessary before a weed population becomes resistant. These include:

- *Seedbank life.* Resistance is slower to appear in weed species that have higher levels of seed dormancy. While the seed produced after each application of herbicide may contain a higher proportion of resistant individuals, susceptible seed from the seedbank will dilute resistance levels.

- *Fitness of resistant biotypes.* In some instances herbicide resistant weeds may be less vigorous than susceptible plants of the same species. The ability of the weed to compete with other plants and set seed may therefore be reduced. Development of resistance may be slower where there is a significant fitness penalty associated with the resistance mechanism. For example, triazine (eg atrazine) resistance has a fitness penalty because the resistance mechanism involves a mutation in photosynthesis, the engine for plant growth. Hence, triazine-tolerant canola varieties have a lower yield potential compared with conventional lines.
- *Seed production.* The greater the number of seeds produced by a resistant plant, the greater the number of resistant plants that will need to be controlled in the following year. Annual ryegrass can produce up to 80,000 seeds/m<sup>2</sup> and wild radish and charlock around 30,000 seeds/m<sup>2</sup>.
- *Importation of resistance.* It is possible for resistance to be introduced into a weed population, although the impact it has will depend on the weed numbers involved. Introduction can be the result of various seed dispersal mechanisms; resistant seed in stock feeds, hay, crop seed, machinery, soil or animal movement. This is particularly important with rare forms such as glyphosate resistance.
- *Chance.* The distribution of resistant individuals within a population is not uniform. On average, all annual ryegrass populations start off with about 1 plant in 17,000 with resistance to Group B herbicides. In reality, some populations have 1 plant in 8,000, and others 1 in 100,000, purely as a function of chance.

## Herbicide resistance in Australia

Throughout the world herbicide resistance is an increasing problem. Information compiled by Dr Ian Heap at [www.weedscience.org/in.asp](http://www.weedscience.org/in.asp) provides details of global and Australian herbicide resistant weeds.

Worldwide, more weed species have developed resistance to Group B herbicides than to any other MOA group. A large number of grass (Table HR5) and broadleaf (Table HR6) weed species have populations which have been confirmed to be resistant to a range of herbicides across Australia.

**Table HR5** Known populations of herbicide resistant grass weeds in Australia (compiled by Stewart 2005)

Weed species	Herbicide group	Example herbicide	States with confirmed resistant populations					
			WA	SA	Vic	NSW	Tas	Qld
Annual ryegrass ( <i>Lolium rigidum</i> )	A – ‘fops’	diclofop	X	X	X	X	X	
	A – ‘dims’	sethoxydim	X	X	X	X	X	
	B – sulfonyleureas	chlorsulfuron	X	X	X	X		
	B – imidazolinones	imazapic, imazapyr	X	X	X	X		
	C – triazines	simazine, atrazine	X	X	X			
	C – substituted ureas	diuron		X	X			
	D – dinitroanilines	trifluralin	X	X	X	X		
	Q – triazoles	amitrole	X	X				
	M – glycines	glyphosate	X	X	X	X		
Barley grass ( <i>Hordeum leporinum</i> ; <i>Hordeum glaucum</i> )	A – ‘fops’	haloxyfop, fluazifop		X	X	X		
	A – ‘dims’	sethoxydim		X				
	L – bipyridiliums	paraquat		X	X		X	
	B – sulfonyleureas	sulfosulfuron, sulfometuron	X					
Brome grass ( <i>Bromus diandrus</i> )	A – ‘fops’	haloxyfop			X			
Barnyard grass ( <i>Echinochloa colona</i> )	C – triazines	atrazine				X		
	M – glycines	glyphosate				X		
Brome grass ( <i>Bromus rigidus</i> )	A – ‘fops’	quizalofop	X					
Giant Parramatta grass ( <i>Sporobolus fertilis</i> )	J – alkanolic acids	fluproponate				X		
Crabgrass ( <i>Digitaria sanguinalis</i> )	A – ‘fops’	fluazifop, haloxyfop	X	X				
	B – imidazolinones	imazethapyr		X				
Liverseed grass ( <i>Urochloa panicoides</i> )	C – triazines	atrazine						X
Paradoxa grass ( <i>Phalaris paradoxa</i> )	A – ‘fops’	fluazifop				X		
	A – ‘dims’	sethoxydim				X		
Serrated tussock ( <i>Nasella trichotoma</i> )	J – alkanolic acids	fluproponate			X			
Silver grass ( <i>Vulpia</i> spp.)	L – bipyridiliums	paraquat			X			
Wild oat ( <i>Avena</i> spp.)	A – ‘fops’	diclofop	X	X	X	X		X
	A – ‘dims’	tralkoxydim	X	X	X	X		X
	B – sulfonyleureas	iodosulfuron-methyl-sodium		X				
	Z – arylaminopropionic acids	flamprop-methyl				X		

Note: Collated from information presented at [www.weedscience.org/in.asp](http://www.weedscience.org/in.asp) and other published literature.

**Table HR6** Known populations of herbicide resistant broadleaf weeds in Australia (compiled by Stewart 2005)

Weed species	Herbicide group	Example herbicide	States with confirmed resistant populations						
			WA	SA	Vic	NSW	Tas	Qld	
African turnip weed ( <i>Sisymbrium thellungi</i> )	B – sulfonyleureas	chlorsulfuron							X
Black bindweed ( <i>Fallopia convolvulus</i> )	B – sulfonyleureas	chlorsulfuron							X
Calomba daisy ( <i>Pentzia suffruticosa</i> )	B – sulfonyleureas	metosulfuron-methyl		X					
Capeweed ( <i>Arctotheca calendula</i> )	L – bipyridiliums	paraquat, diquat			X				
Charlock ( <i>Sinapis arvensis</i> )	B – sulfonyleureas	chlorsulfuron				X			
Common sowthistle ( <i>Sonchus oleraceus</i> )	B – sulfonyleureas	chlorsulfuron				X			X
Dense-flowered fumitory ( <i>Fumaria densiflora</i> )	D – dinitroanilines	trifluralin		X		X			
Indian hedge mustard ( <i>Sisymbrium orientale</i> )	B – sulfonyleureas	chlorsulfuron	X	X		X			X
	B – sulfonamides	metosulam	X	X		X			X
	B – imidazolinones	imazethapyr		X					
	I – phenoxies	2,4-D		X					
Paterson's curse / Salvation Jane ( <i>Echium plantagineum</i> )	B – sulfonyleureas	chlorsulfuron	X	X					
	B – sulfonamides	metosulam	X	X					
Prickly lettuce ( <i>Lactuca serriola</i> )	B – sulfonyleureas	triasulfuron		X					
	B – imidazolinones	imazethapyr		X					
Sand rocket ( <i>Diplotaxis tenuifolia</i> )	B – sulfonyleureas	chlorsulfuron		X					
Stinging nettle ( <i>Urtica urens</i> )	C – triazines	simazine, atrazine			X				
Turnip weed ( <i>Rapistrum rugosum</i> )	B – sulfonyleureas	chlorsulfuron				X			X
Wild radish ( <i>Raphanus raphanistrum</i> )	B – sulfonyleureas	chlorsulfuron	X	X	X	X			
	B – sulfonamides	metosulam	X	X					
	B – imidazolinones	imazapic, imazapyr	X	X					
	C – triazines	simazine, atrazine	X						
	C – triazinones	metribuzin	X						
	F – nicotinanalides	diflufenican	X						
	I – phenoxies	2,4-D	X						
Wild turnip ( <i>Brassica tournefortii</i> )	B – sulfonyleureas	chlorsulfuron	X	X					
	B – sulfonamides	metosulam	X						

Note: Collated from information presented at [www.weedscience.org/in.asp](http://www.weedscience.org/in.asp) and other published literature.

## Extent of resistance to selective herbicides in Australia

The Western Australian Herbicide Resistance Initiative (WAHRI) conducted a wide-scale survey of 264 cropping paddocks across the Western Australian wheat belt in 1999 to identify the number of herbicide resistant annual ryegrass populations.

Of the populations surveyed, 46% were found to be resistant to diclofop-methyl (Group A 'fop') and 64% to chlorsulfuron (Group B 'SU'). Multiple resistance to diclofop-methyl and chlorsulfuron was detected in 37% of the populations. Only 28% were susceptible to both herbicides (Llewellyn and Powles 2001).

In 2003 WAHRI collected seed of 90 wild radish populations from 500 surveyed paddocks across the Western Australian wheat belt. Screening of these populations in 2004 found that 60% contained plants resistant to chlorsulfuron. In addition, 6% were resistant to atrazine (Group C) with 68% developing resistance, and 5% resistant to 2,4-D (Group I) with 62% developing resistance. Over 60% of the populations had resistance to two herbicides (Walsh et al 2005).

While there were regional differences in the levels of resistance detected, the results of these surveys highlight the scope of the problem of herbicide resistant annual ryegrass and wild radish in annual cropping regions in Western Australia.

In a similar survey of annual ryegrass populations conducted in South Australia in 1998 and 2003 (Table HR7), the level of Group A resistance doubled, that of Group B resistance trebled and multiple resistance increased ten-fold in 5 years (Preston 2004).

A survey of 185 annual ryegrass populations collected (Preston 2004) at random in the Mid North and Upper Yorke Peninsula of South Australia in 2003 found moderate Group D resistance in up to half of the populations and high levels of Group D resistance in 14% (Table HR8). These populations were also found to have high levels of resistance to Group A and B herbicides (Boutsalis et al 2006).

A recent survey of paddocks in northern New South Wales and Queensland identified approximately 10% that had wild oat populations resistant to Group A 'fop' herbicides. A small number of paddocks had Group A resistant annual ryegrass and several had Group B resistant common sowthistle, turnip weed, African turnip weed and charlock. One paddock had Group C resistant barnyard grass (Widderick and Galea 2004).

**Table HR7** Survey results from South Australia in 1998 and 2003 showing significant increases in the levels of Group A, Group B and multiple resistance (Preston 2004)

Herbicide group	Resistance in annual ryegrass populations in SA (%)	
	1998 (215 samples)	2003 (170 samples)
Group A herbicides	38	76
Group B herbicides	21	75
Group A and Group B herbicides	6	59

**Table HR8** Results of herbicide resistance in 185 randomly collected populations of annual ryegrass from the Mid North and Upper Yorke Peninsula in 2003 – data shows the percentage of samples with > 20% resistant individuals (measured as % survival) (Boutsalis unpublished)

	MOA group				
	A – 'fop'	A – 'dim'	B	D	D
Herbicide	diclofop	tralkoxydim	chlorsulfuron	trifluralin	trifluralin
Rate per ha	1,000 mL	500 g	20 g	500 mL	1,000 mL
% survival	77	45	75	49	14

## Herbicide resistance case study #2

Multiple resistance in wild radish

**Weed:** Two wild radish populations collected from the northern wheat belt, Western Australia

**Rotation:** Previous 17 seasons of intensive wheat/lupin, with two herbicide applications per year

**Herbicide use history:** 1983–99 (Table HR9)

**Resistance profile:** Population 1 – resistant to:  
 Group I phenoxyes (2,4-D)  
 Group F nicotinanalides (diflufenican)  
 Group C triazinones (metribuzin) and triazines (atrazine)  
 Population 2 – resistant to:  
 Group B sulfonylureas (chlorsulfuron), imidazolinones (imazethapyr) and sulfonamides (metosulam)  
 Group I phenoxyes (2,4-D)  
 Group F nicotinanalides (diflufenican)

The case study is situated in sand-plain country which harbours large populations of very vigorous wild radish. As seen in Table HR9, the high-risk Group B herbicides were heavily used.

Lupin crops received simazine (Group C) plus atrazine (Group C) in most years, with diflufenican (Group F) also being used as a post-emergent in later years. Wheat predominantly received triasulfuron (Group B) as a pre-emergent, followed by 2,4-D amine (Group I) as a post-emergent. Early herbicide application followed by a second 'lower risk' MOA group would have slowed development of resistance for some years.

Because crop competition was not actively implemented (lupins are poor competitors), herbicides were the sole weed control tactic in this rotation. It is unlikely that any tactics for reduction of seed-set were implemented over this 17-year period. The wild radish population expanded massively towards the end of the case study period, implying that large numbers of weeds were treated with herbicide each season. Such practice further increased the risk for developing resistance.

The two populations of wild radish in this case study are now resistant to three MOA groups. Although the herbicide MOA was rotated, seed-set was not prevented in weeds that survived herbicide applications, and multiple resistance therefore developed. Population 1 developed target-site cross-resistance to metribuzin from the application of triazine herbicides.

Multiple resistance now forces the grower to use more expensive techniques to control wild radish, reducing returns from affected paddocks. Tactics may include restricting the use of lupin or other broadleaf crops, wide-row sowing and shielded inter-row spraying of Spray.Seed®, crop-topping (yield reducing) and long chemical fallow.

### Extent of resistance to non-selective herbicides in Australia

In 1996 glyphosate resistance was identified for the first time, confirmed in annual ryegrass in Australia (Heap 2006). Since then more than 40 populations of glyphosate resistant annual ryegrass have been identified across Australia. In addition, there is confirmed resistance to paraquat + diquat (eg Spray.Seed®) in populations of barley grass, vulpia and capeweed.

As with all other herbicides at risk of evolving resistant weed populations, selection for glyphosate resistance

**Table HR9** Number of herbicide applications for two wild radish weed populations in Western Australian herbicide resistance case study #2 (Walsh et al 2003)

MOA group	Herbicide	Number of applications 1983–99	
		Population 1	Population 2
B ('SU')	triasulfuron	9	8
	metsulfuron-methyl	1	0
	chlorsulfuron	1	0
C (triazine)	simazine	8	9
	atrazine	4	4
F (nicotinanalide)	diflufenican	4	5
I (phenoxy)	2,4-D (amine +/- or ester)	9	8
M	glyphosate	9	9

is promoted by particular management activities (Table HR10). It is important to avoid 'risk-increasing' actions and include 'risk-decreasing' tactics.

**Table HR10** Factors that influence the risk of glyphosate resistance evolving in annual ryegrass (Glyphosate Sustainability Working Group 2006)

Risk-increasing actions	Risk-decreasing actions
<ul style="list-style-type: none"> <li>• continuous reliance on glyphosate pre-seeding</li> <li>• lack of tillage</li> <li>• lack of effective in-crop weed control</li> <li>• frequent glyphosate-based chemical fallow</li> <li>• inter-row glyphosate use (unregistered)</li> <li>• frequent crop-topping with glyphosate</li> <li>• high weed numbers</li> </ul>	<ul style="list-style-type: none"> <li>• the double knock technique*</li> <li>• strategic use of alternative knockdown groups</li> <li>• full-cut cultivation at sowing</li> <li>• effective in-crop weed control</li> <li>• use of alternative herbicide groups or tillage for inter-row and fallow weed control</li> <li>• non-herbicide practices for weed seed kill</li> <li>• crop-topping with alternative herbicide groups</li> <li>• farm hygiene to prevent resistant seed movement</li> </ul>

\*The double knock technique is defined as using a full-disturbance cultivation OR the full label rate of a paraquat-based product (Group L herbicide) following the glyphosate (Group M herbicide) knockdown application.

Knockdown herbicides are a critical weed management tool in our current farming systems. As with all weed control tactics, non-selective herbicides should always be used in a planned program of weed management in conjunction with a number of other tactics from different tactic groups.

### Herbicide resistance case study #3

Glyphosate resistance in annual ryegrass

**Weed:** A population of annual ryegrass collected from the Liverpool Plains, New South Wales, in 1999

**Rotation:** Conventionally sown to sorghum, wheat and sunflowers between 1981 and 1989. From 1990 to 1998 no-till wheat – long fallow – sorghum was introduced, and glyphosate was used as the sole weed control in fallows

**Herbicide use history:** 1981–99 (Table HR11)

**Resistance profile:** The high number of glyphosate applications (Table HR11) on this population resulted in the evolution of glyphosate resistance; the population was not resistant to Group A herbicides

This resistance profile is typical of that for minimum-tillage growers on vertisol soils in northern New South Wales, where wild oats represent the major annual grass weed. Annual ryegrass only became a major weed of winter crops and fallows after the introduction of minimum-tillage systems.

Crop competition was not actively implemented in this case study. Introduction of no-till systems reduced crop competition, as did increased row spacings (widened to 38 cm for wheat). Post-emergent grass herbicides were used to target wild oat control.

During the 1990s the problem of rising watertables and the threat of atrazine (Group C) residues in groundwater were highlighted to growers. The concept of 'opportunity cropping' (sowing the most suitable crop when the soil profile contains 1 m water) was promoted. On alkaline soils this practice excluded the effective use of herbicides with long residuals such as the sulfonylureas (Group B), including chlorsulfuron and triasulfuron. The use of atrazine in winter fallows also declined although atrazine was an effective pre-emergent control for annual ryegrass in winter fallows before sorghum.

This decline in use of residual herbicides created a heavy reliance on glyphosate to control fallow weed populations.

**Table HR11** Number of herbicide applications for New South Wales herbicide resistance case study #3 (Storrie and Cook 2002)

MOA group	Herbicide	Number of applications 1981–99
M	glyphosate	20
A (fop)	fenoxaprop-p-ethyl	1
	quizalofop-p-ethyl	1
	clodinafop-propargyl	1
B ('SU')	chlorsulfuron	1
	metsulfuron-methyl	1
	thifensulfuron-methyl	1
C (triazine)	atrazine	2
I (phenoxy)	2,4-D (amine +/- or ester)	6
	MCPA	2
I (pyridine)	picloram	2
	fluroxypyr	3

No tactics from Tactic Group 1 (Deplete weed seed in the target area soil seedbank), Tactic Group 3 (Stop weed seed-set), or Tactic Group 4 (Prevent viable weed seeds within the target area being added to the soil seedbank) were used.

Growers and agronomists were not monitoring levels of weed control or changes in weed species and number, so no risk reducing measures were taken until resistance was suspected.

## Glyphosate Sustainability Working Group

The national Glyphosate Sustainability Working Group is a collaborative initiative involving research, industry and extension representatives, with the purpose of promoting the sustainable use of glyphosate in Australian agriculture.

### Priority goals

1. Increase the sustainability of glyphosate usage through the development and delivery of clear and consistent information based on industry consensus.
2. Increase collaboration and consistency among the glyphosate research and extension activities of key research, extension and industry groups.
3. Contribute to the development of research, development and extension initiatives aimed at improving the management of glyphosate.

### Key activities

The Glyphosate Sustainability Working Group's website, hosted by the Cooperative Research Centre for Australian Weed Management ([www.weeds.crc.org.au/glyphosate](http://www.weeds.crc.org.au/glyphosate)), is used as the main method of information exchange.

The group has developed a simple list of factors that have an influence on the risk of annual ryegrass populations developing resistance to glyphosate (Table HR10, available as a poster on the website).

There is also an active register containing information about all the known glyphosate resistant weed populations present in Australia. Populations are added to the register after confirmation by one of the testing services or researchers.

## CropLife Australia Ltd Herbicide Resistance Management Committee

CropLife Australia Ltd (formerly Avcare – The National Association for Crop Production and Animal Health) has developed a series of Resistance Management Strategies ([www.croplifeaustralia.org.au](http://www.croplifeaustralia.org.au)) for herbicides from most MOA groups. The specific guidelines for the use of crop protection products are designed to reduce the selection pressure for resistance.

Development and implementation of an IWM plan, incorporating tactics from a number of tactic groups (see *Section 4: Tactics for managing weed populations*) and following the recommendations in the CropLife Australia Ltd Resistance Management Strategies, can extend the effective life of herbicides in crop paddocks and assist management of herbicide resistant weed populations.

## Weed species at risk

A wide range of crop weeds in Australia have populations confirmed to be resistant to a range of herbicide MOA groups (Tables HR5 and HR6).

Global examples of herbicide resistance are presented in Table HR12. Although these weeds are present in Australia, to date no populations of the herbicide resistances shown in this table have been reported here.

In Australia's northern grain region, populations of 10 species of weeds have been confirmed as herbicide resistant and more have been identified as at risk of developing resistance, particularly to glyphosate. Apart from common sowthistle and summer grasses, the weed threats differ across the northern region according to the farming system used.

It is mostly winter weeds that are at greatest risk in central New South Wales, whereas a mix of both summer and winter weeds are at risk in northern New South Wales and southern Queensland. Summer weeds are at the greatest risk of developing resistance in central Queensland (Walker et al 2004).

Summer weeds include sweet summer grass, barnyard grass, liverseed grass, common sowthistle and parthenium weed. The risk for winter weeds is mainly expansion of current known problems such as glyphosate resistance in annual ryegrass, Group B resistance in brassica weeds and Group A resistance in wild oats. Glyphosate and Group B resistance in wild oats are also considered a risk.

The extensive use of trifluralin (Group D) in no-till farming systems in southern Australia is considered a high risk for resistance in annual ryegrass. Shepherd's purse and brome grass are also at risk of developing Group B resistance.

While there are no populations of annual ryegrass identified as resistant to paraquat in Australia, there is resistance to paraquat in South African vineyards. Paraquat (Group L) resistance was also confirmed in weed populations in *Conyza bonariensis* (1989) in Egypt and *Poa annua* (1981) in the United Kingdom (Heap 2006).

**Table HR12** Resistance watch: confirmed resistances in overseas populations of common weed species in crops (compiled by Stewart, Walker and Storrie 2005)

Weed species	Herbicide group	Example herbicide	Countries with confirmed resistant populations
Annual ryegrass ( <i>Lolium rigidum</i> )	L – bipyridiliums	paraquat	South Africa
Ball mustard ( <i>Neslia paniculata</i> )	B – sulfonylureas	metsulfuron-methyl	Canada
Barnyard grass ( <i>Echinochloa</i> spp.)	A – ‘fops’ B – imidazolinones C – acetamides D – dinitroanilines J – thiocarbamates	fenoxaprop, quizalofop imazethapyr propanil pendimethalin molinate	Thailand, USA Yugoslavia USA, Greece, Italy, Thailand Bulgaria USA, China
Brome grass ( <i>Bromus</i> spp.)	B – sulfonylureas B – imidazolinones C – triazines C – substituted ureas	sulfosulfuron imazamox atrazine chlorotoluron	USA USA France, Spain Spain
Charlock ( <i>Sinapis arvensis</i> )	B – imidazolinones C – triazines C – triazinones I – phenoxies I – pyridines I – benzoic acids	imazethapyr atrazine metribuzin 2,4-D picloram dicamba	USA Canada Canada Canada Canada Canada
Common chickweed ( <i>Stellaria media</i> )	B – sulfonylureas  C – triazines I – phenoxies	chlorsulfuron  atrazine mecoprop	Canada, Denmark, Ireland, New Zealand, Norway, South Africa, Sweden, United Kingdom Germany United Kingdom
Crabgrass ( <i>Digitaria sanguinalis</i> )	C – triazines	atrazine	France, Poland
Crabgrass ( <i>Digitaria ciliaris</i> )	A – ‘fops’	fluazifop-p	Brazil
Crowsfoot grass ( <i>Eleusine indica</i> )	A – ‘fops’ B – imidazolinones D – dinitroanilines L – bipyridiliums M – glycines	fluazifop-butyl imazapyr trifluralin paraquat glyphosate	Brazil, Malaysia Costa Rica USA Malaysia, USA Malaysia
Fleabane ( <i>Conyza</i> spp.)	B – sulfonylureas C – triazines  C – substituted ureas L – bipyridiliums  M – glycines	chlorsulfuron atrazine  linuron paraquat  glyphosate	Israel, Poland, USA Israel, Spain, Belgium, Czech Republic, France, Poland, Switzerland, United Kingdom, USA France, USA Egypt, Japan, Malaysia, Sri Lanka, Taiwan, South Africa, Belgium, Canada, USA USA, South Africa, Spain
Lesser canary grass ( <i>Phalaris minor</i> )	A – ‘fops’ B – sulfonylureas  C – substituted ureas	fenoxaprop sulfosulfuron, iodosulfuron-methyl, mesosulfuron isoproturon	Mexico, Israel, USA, South Africa South Africa India
Paradoxa grass ( <i>Phalaris paradoxa</i> )	C – triazines	atrazine	Israel
Shepherd’s purse ( <i>Capsella bursa-pastoris</i> )	C – triazines	atrazine	Poland

Weed species	Herbicide group	Example herbicide	Countries with confirmed resistant populations
Wild oat ( <i>Avena</i> spp.)	B – sulfonylureas	iodosulfuron-methyl, rimsulfuron	South Africa, Canada, United Kingdom, USA
	J – thiocarbamates	tri-allate	Canada, USA
	D – benzamides	propyzamide	USA
Wireweed ( <i>Polygonum aviculare</i> )	C – triazines	atrazine	Belgium, Netherlands
	Q – triazoles	amitrole	Belgium

Note: Collated from information presented at [www.weedscience.org/in.asp](http://www.weedscience.org/in.asp) and other published literature.

## Herbicide resistance testing

Testing herbicide resistance status provides essential information about weed populations for planning both weed management and enterprise sequence.

When a spray failure occurs it is essential to determine if the failure was due to resistance. This can avoid the unnecessary use of ineffective herbicides that are unable to kill the weeds in question, optimise crop yield and provide essential information on in-crop and future weed management.

Testing can determine which herbicides will work in the current or next season. For example, annual ryegrass may not be controlled by diclofop-methyl (Group A 'fop') but may still be susceptible to tralkoxydim (Group A 'dim'), which allows some flexibility with cereal crops.



Effect of trifluralin (400 g ai/ha) 6 weeks after treatment. The front three pots are known annual ryegrass standards – from left to right are L: susceptible, M: trifluralin resistant biotype with intermediate resistance, R: trifluralin resistant biotype with strong resistance. Pots in the background represent randomly collected annual ryegrass samples from Victoria in 2005 (Mallee and Wimmera regions). A pot test was conducted in winter 2006. Each pot represents seed collected from one paddock.

Photo: Peter Boutsalis

Knowing which herbicides are still effective will allow future planning of enterprise sequence and help determine which cultural management techniques must be employed.

Testing can be conducted in situ or by a commercial testing service. In-situ tests provide a good visual identification of resistance for growers, but can be more difficult to interpret due to variable paddock conditions and the increasing size of weeds before they can be re-treated.

For information on how to test for resistance, see *Section 5: Implementing an IWM program using tactic groups* and the Glyphosate Sustainability Working Group website ([www.weeds.crc.org.au/glyphosate/index.html](http://www.weeds.crc.org.au/glyphosate/index.html)).

## The future

History has shown that the trend for increasing herbicide resistance in Australian cropping systems is likely to continue, at least in the near future. Due to the great success of herbicides improving weed control and farmer returns over the last 30 years, non-herbicide management has been forgotten by the majority of advisors and growers.

Herbicide resistance is the impetus for the re-learning of integrated weed management. Growers in more favourable climatic areas have more options available and better cash flows to fund necessary changes in management. Growers in drier areas, however, face greater challenges in managing highly variable seasonal conditions and cash flows, which determine their ability to adopt and implement change. Convincing growers to introduce changes in weed management sooner rather than later is a challenging and long-term task for all farm advisors.

**Table HR13** Herbicides classified according to primary mode-of-action (MOA) group

Herbicide subgroup	Herbicide active ingredient	Example products
<b>Group A – Inhibitors of acetyl coA carboxylase (Inhibitors of fat synthesis/ACC'ase inhibitors)</b>		
<i>Aryloxyphenoxypropionates – 'fops'</i>	clodinafop cyhalofop diclofop fenoxaprop fluazifop haloxyfop propaquizafop quizalofop	Topik® Barnstorm® Cheetah® Gold*, Decision®*, Hoegrass®, Tristar® Advance* Cheetah® Gold*, Tristar® Advance*, Wildcat® Fusilade®, Fusion®* Motsa®*, Verdict® Correct® Targa®
<i>Cyclohexanediones – 'dims'</i>	butroxydim clethodim sethoxydim tepraloxym tralkoxydim	Factor®, Fusion®* Motsa®*, Select® Cheetah® Gold*, Decision®*, Sertin® Aramo® Achieve®
<i>'Fops' + 'dims'</i>	fluazifop+butroxydim haloxyfop+clethodim diclofop-fenoxaprop+sethoxydim diclofop-methyl+sethoxydim	Fusion® Motsa® Cheetah® Gold* Decision®
<i>Phenylpyrazoles – 'dens'</i>	pinoxaden	Axial®
<b>Group B – Inhibitors of acetolactate synthase (ALS inhibitors)</b>		
<i>Sulfonylureas – 'SUs'</i>	azimsulfuron bensulfuron chlorsulfuron ethametsulfuron halosulfuron iodosulfuron mesosulfuron metsulfuron rimsulfuron sulfometuron sulfosulfuron thifensulfuron triasulfuron tribenuron trifloxysulfuron	Gulliver® Londax® Glean® Bounty®* Sempra® Hussar® Atlantis® Ally®, Harmony®* M, Crossbow®*, Trounce®* Titus® Oust® Monza® Harmony®* M Logran®, Logran® B-Power* Express® Envoke®, Krismat®*
<i>Imidazolinones – 'Imis'</i>	imazamox imazapic imazapyr imazethapyr	Raptor®, Intervix®* Flame®, Midas®*, OnDuty®* Arsenal®, Midas®*, OnDuty®*, Intervix®*, Lightning®* Spinnaker®, Lightning®*
<i>Triazolopyrimidines – Sulfonamides</i>	flumetsulam metosulam florasulam pyroxulam	Broadstrike® Eclipse® Torpedo®*, X-Pand®* Crusader®
<i>Pyrimidinylthiobenzoates</i>	pyrithiobac-Na	Staple®
<b>Group C – Inhibitors of photosynthesis at photosystem II (PS II inhibitors)</b>		
<i>Triazines</i>	ametryn atrazine cyanazine prometryn simazine terbutryn	Primatol® Z, Gesapax® Combi*, Krismat® Gesaprim®, Gesapax® Combi*, Primextra® Gold* Bladex® Gesagard®, Cotogard®*, Bandit®* Gesatop® Igran®, Agtryne® MA*
<i>Triazinones</i>	hexazinone metribuzin	Velpar® L, Velpar® K4* Sencor®

Herbicide subgroup	Herbicide active ingredient	Example products
<i>Ureas</i>	diuron	Karmex <sup>®</sup> , Krovar <sup>®*</sup> , Velpar <sup>®</sup> K4*
	fluometuron	Cotoran <sup>®</sup> , Cotogard <sup>®*</sup> , Bandit <sup>®*</sup>
	linuron	Afalon <sup>®</sup>
	methabenzthiazuron	Tribunil <sup>®</sup>
	siduron	Tupersan <sup>®</sup>
	tebuthiuron	Graslan <sup>®</sup>
<i>Nitriles</i>	bromoxynil	Buctril <sup>®</sup> , Buctril <sup>®</sup> MA*, Barrel <sup>®*</sup> , Jaguar <sup>®*</sup>
	ioxynil	Totril <sup>®</sup> , Actril DS*
<i>Phenylcarbamates</i>	phenmedipham	Betanal <sup>®</sup>
<i>Pyridazinones</i>	chloridazon	Pyramin <sup>®</sup>
<i>Amides</i>	propanil	Stam <sup>®</sup>
<i>Benzothiadiazinones</i>	bentazone	Basagran <sup>®</sup> , Basagran <sup>®</sup> M60*
<i>Uracils</i>	bromacil	Hyvar <sup>®</sup> , Krovar <sup>®*</sup>
	terbacil	Sinbar <sup>®</sup>
<b>Group D – Inhibitors of microtubule assembly</b>		
<i>Dinitroanilines – DNAs</i>	oryzalin	Surflan <sup>®</sup> , Yield <sup>®*</sup>
	pendimethalin	Stomp <sup>®</sup>
	trifluralin	Treflan <sup>®</sup> , Yield <sup>®*</sup>
<i>Benzoic acids</i>	chlorthal-dimethyl	Dacthal <sup>®</sup> , Prothal <sup>®*</sup>
<i>Benzamides</i>	propyzamide	Kerb <sup>®</sup>
<b>Group E – Inhibitors of mitosis / microtubule organisation</b>		
<i>Carbamates</i>	chlorpropham	Chlorpropham <sup>®</sup>
<b>Group F – Bleachers: Inhibitors of carotenoid biosynthesis at the phytoene desaturase step (PDS inhibitors)</b>		
<i>Nicotinilides</i>	diflufenican	Brodal <sup>®</sup> , Jaguar <sup>®*</sup> , Tigrex <sup>®*</sup> , Chipco Spearhead <sup>®*</sup>
<i>Picolinamides</i>	picolinafen	Paragon <sup>®*</sup> , Sniper <sup>®</sup>
<i>Pyridazinones</i>	norflurazon	Solicam <sup>®</sup>
<b>Group G – Inhibitors of protoporphyrinogen oxidase (PPOs)</b>		
<i>Diphenylethers</i>	acifluorfen	Blazer <sup>®</sup>
	oxyfluorfen	Goal <sup>®</sup>
<i>N-phenylphthalimides</i>	flumioxazin	Pledge <sup>®</sup>
	flumiclorac	Resource <sup>®</sup>
<i>Oxadiazoles</i>	oxadiazon	Ronstar <sup>®</sup>
<i>Phenylpyrazoles</i>	pyraflufen	Ecopar <sup>®</sup>
<i>Pyrimidindiones</i>	butafenacil	Logran <sup>®</sup> B-Power*
<i>Triazolinones</i>	carfentrazone	Affinity <sup>®</sup>
<b>Group H – Bleachers: Inhibitors of 4-hydroxyphenyl-pyruvate dioxygenase (HPPDs)</b>		
<i>Isoxazoles</i>	isoxaflutole	Balance <sup>®</sup>
<i>Pyrazoles</i>	benzofenap	Taipan <sup>®</sup> , Viper <sup>®</sup>
	pyrasulfotole	Precept <sup>®*</sup>
<b>Group I – Disrupters of plant cell growth</b>		
<i>Phenoxyacetic acids – Phenoxy</i>	2,4-D	Amicide <sup>®</sup> , Actril DS <sup>®*</sup>
	2,4-DB	Trifolamine <sup>®</sup>
	MCPA	MCPA, Buctril <sup>®</sup> MA*, Banvel M <sup>®*</sup> , Midas <sup>®*</sup> , Paragon <sup>®*</sup> , Tigrex <sup>®*</sup> , Barrel <sup>®*</sup> , Tordon <sup>®</sup> 242*, Basagran <sup>®</sup> M60*, Chipco Spearhead <sup>®*</sup> , Agtryne <sup>®</sup> MA*
	mecoprop	Mecopropamine <sup>®</sup> , Mecoban <sup>®</sup> , Methar <sup>®</sup> , Tri-Kombi <sup>®*</sup>
<i>Benzoic acids</i>	dicamba	Banvel <sup>®</sup> , Banvel <sup>®</sup> M*, Barrel <sup>®*</sup> , Mecoban <sup>®</sup> Methar Tri-Kombi <sup>®*</sup>

Herbicide subgroup	Herbicide active ingredient	Example products
<i>Pyridine carboxylic Acids – Pyridines</i>	aminopyralid clopyralid fluroxypyr picloram triclopyr	Hotshot®* Lontrel®, Torpedo®*, Chipco Spearhead®* Starane®, Hotshot®* Tordon®, Tordon® 242*, Crossbow®*, Grazon®* Garlon®, Grazon®*
<i>Quinoline carboxylic acids</i>	quinclorac	Drive®
<b>Group J – Inhibitors of fat synthesis (not ACCase inhibitors)</b>		
<i>Chlorocarboxylic acids</i>	2,2-DPA flupropanate	Dalapon® Frenock®
<i>Thiocarbamates</i>	EPTC molinate pebulate prosulfocarb thiobencarb triallate	Eptam® Ordram® Tillam® Boxer® Gold* Saturn® Avadex®
<i>Phosphorodithioates</i>	bensulide	Prefar®
<i>Benzofurans</i>	ethofumesate	Tramat®
<b>Group K – Inhibitors of cell division / Inhibitors of very long chain fatty acids (VLCFA inhibitors)</b>		
<i>Acetamides</i>	napropamide	Devrinol®
<i>Chloroacetamides</i>	metolachlor propachlor	Dual® Gold, Primextra® Gold*, Boxer® Gold* Ramrod®, Prothal®*
<b>Group L – Inhibitors of photosynthesis at photosystem I (PSI inhibitors)</b>		
<i>Bipyridils</i>	diquat paraquat	Reglone®, Spray Seed®* Gramoxone®, Spray Seed®*
<b>Group M – Inhibitors of EPSP synthase</b>		
<i>Glycines</i>	glyphosate	Roundup®, Trounce®*, Illico®*
<b>Group N – Inhibitors of glutamine synthetase</b>		
<i>Phosphinic acids</i>	glufosinate	Basta®, Liberty®
<b>Group O – Inhibitors of cell wall (cellulose) synthesis</b>		
<i>Nitriles</i>	dichlobenil	Casoron®
<i>Benzamides</i>	isoxaben	Gallery®, X-Pand®*
<b>Group P – Inhibitors of auxin transport</b>		
<i>Phthalamates</i>	naptalam	Alanap-L®
<b>Group Q – Bleachers: Inhibitors of carotenoid biosynthesis unknown target</b>		
<i>Triazoles</i>	amitrole	Amitrole® T, Illico®*
<i>Isoxazolidinones</i>	clomazone	Command®, Viper®
<b>Group R – Inhibitors of dihydropteroate synthase (DHP inhibitors)</b>		
<i>Carbamates</i>	asulam	Asulox®
<b>Group Z – Herbicides with unknown and probably diverse sites of action</b>		
<i>Arylamino propionic acids</i>	flamprop	Mataven® L
<i>Dicarboxylic acids</i>	endothal	Endothal®
<i>Organoarsenicals</i>	DSMA (disodium methylarsonate) MSMA	Methar® Daconate®

Note: This table is based on the revised mode of action groups from CropLife Australia as at 28 February 2008. The use of brand, trade and proprietary names is solely for the purpose of assisting users in identifying products. It does not imply a preferred recommendation. Alternative products containing the same active ingredient may perform similarly to those products specified.

\* This product contains more than one active constituent

## Contributors

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