SECTION 2
HERBICIDE RESISTANCE

INTEGRATED WEED MANAGEMENT IN AUSTRALIAN CROPPING SYSTEMS

SECTION 2: HERBICIDE RESISTANCE

Herbicide resistant weed populations are now found throughout all cropping areas of Australia. Currently, there are 36 weed species in Australia that have developed resistance to one or more herbicide modes-of-action (MOAs).

The number of herbicide resistant populations and areas affected will continue to increase until integrated weed management practices are widely adopted in Australian cropping systems.

The future

Despite herbicide resistance first being identified in Australia in 1982, growers continue to predominantly rely on herbicides with insufficient focus on seedbank management. The effects of over 20 years of minimum tillage and heavy glyphosate use are only just being expressed in weed populations, with ever increasing numbers being found resistant to glyphosate. Therefore 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 35 years, non-herbicide management has been neglected by many growers.

Herbicide resistance is the impetus for learning integrated weed management. Growers in more favourable climatic areas have more options available and better cash flow to fund necessary changes in management. Growers in drier areas, however, face greater challenges in managing highly variable seasonal conditions and cash flow, which can impact on 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 advisers.

Herbicides

The first modern herbicide was released onto the Australian market in 1946, but it was not until highly effective and low-priced herbicides were released in the late 1970s that herbicides quickly became the most heavily relied upon weed control method for farmers. Even today, despite high use of herbicides leading to high frequencies of resistant weed populations, herbicide control represents the main, and sometimes only, weed management decision made by many farmers.

The widespread adoption of conservation cropping systems has led to an even greater reliance on herbicides due to a corresponding decline in use of alternative weed control methods (such as cultivation). This in turn has resulted in high selection pressure for herbicide resistance in weed populations.

Herbicide resistance facts

- Resistance is the inherited ability of an individual plant to survive and reproduce following a herbicide application that would kill a 'wild type' individual of the same species.
- Thirty-six weed species in Australia currently have populations that are resistant to at least one herbicide mode-of-action (MOA).
- Australian weed populations have developed resistance to 11 distinct MOAs.
- Herbicide resistant individuals are present at very low frequencies in weed populations before the herbicide is first applied.
- The frequency of naturally resistant individuals within a population will vary greatly within and between weed species.
- A weed population is defined as resistant when a herbicide at a label rate that once controlled the population is no longer effective (sometimes an arbitrary figure of 20 per cent survival is used for defining resistance in testing).
- The proportion of herbicide resistant individuals will rise (due to selection pressure) in situations where the same herbicide MOA is applied repeatedly and the survivors are not subsequently controlled.
- Herbicide resistance in weed populations is permanent as long as seed remains viable in the soil. Only weed density can be reduced, not the ratio of resistant to susceptible.

PHOTO: ANDREW STORRIE

Dead (glyphosate-susceptible) annual ryegrass surrounded by glyphosate resistant individuals.

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 herbicidal control. For information on herbicide tolerant crops see *Agronomy 3 Herbicide tolerant (HT) crops* (section 3, page 74).

What is herbicide resistance?

Herbicide resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering or selection of variants produced by tissue culture or mutagenesis.

Herbicide tolerance is the inherent ability of a species to survive and reproduce after herbicide treatment. This implies that there was no selection or genetic manipulation to make the plant tolerant; it is naturally tolerant.

Source: Weed Science Society of America website www.wssa.net/Weeds/Resistance/definitions.htm

Commonly used terms

Herbicide MOA groups

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

MOA groups can be ranked according to the risk of weed populations becoming resistant to those herbicides. Groups A and B are high risk; Groups C to Z are moderate risk. ***There are no low-risk herbicides.***

MOA subgroup chemical classes

Within a herbicide MOA there may be two or more subgroups. With the exception of Group Z, the subgroups are different chemical classes that inhibit the same plant process. There can be differences in efficacy on a species between these subgroups within an MOA. In Group I, for example, 2,4-D (phenoxy subgroup) is highly efficacious on brassica weeds such as mustards and turnips, while dicamba (benzoic subgroup) has low levels of efficacy on this plant family.

There can also be differences in frequency of resistance genes for different subgroups.

Group Z contains herbicides with unknown MOAs.

Selection pressure

Selection pressure is a term used to describe how strongly herbicides select for resistant individuals in a weed population. Every time a herbicide is used, susceptible individuals are killed while resistant individuals survive and produce viable seed. Over time, and with repeated applications of the same herbicide MOA, the population naturally shifts from mostly susceptible to mostly resistant. A high selection pressure herbicide application kills the greatest number of susceptible individuals possible, whereas a low selection pressure spray kills a smaller proportion of the susceptible individuals. These susceptible survivors can then add a higher number of susceptible individuals to the next generation, slowing the overall shift to domination of the population by resistant plants.

Resistance mechanisms

This term is used to describe the specific processes that enable the plant to survive an application of herbicide. Resistance mechanisms are divided into two broad categories so that weed populations may have either target-site or non-target-site based resistance mechanisms or both.

Target-site resistance

Target-site resistance occurs when there is an alteration at the herbicide target site. The alteration occurs at the normal herbicide site of action and is in the form of a structural 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 treatment.

Non-target-site resistance

Non-target-site resistance is used to describe mechanisms other than changes at the target site that 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. For example, populations of wild oats (*Avena* spp.) 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 based resistance.

2 **Across herbicide MOA 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 (*Lolium rigidum*) selected with only Group A herbicides may also be resistant to Group B herbicides. This is usually non-target-site based 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 groups or subgroups. Some populations of resistant annual ryegrass possess both target- and non-target-site resistance to more than one MOA.

How does a weed population develop herbicide resistance?

There are two 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 enables them to survive the application of a particular herbicide that would normally kill this species.

Genetic variation may alter the shape of the target site and/or 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 resistant individuals survive (Figure HR1, below).

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

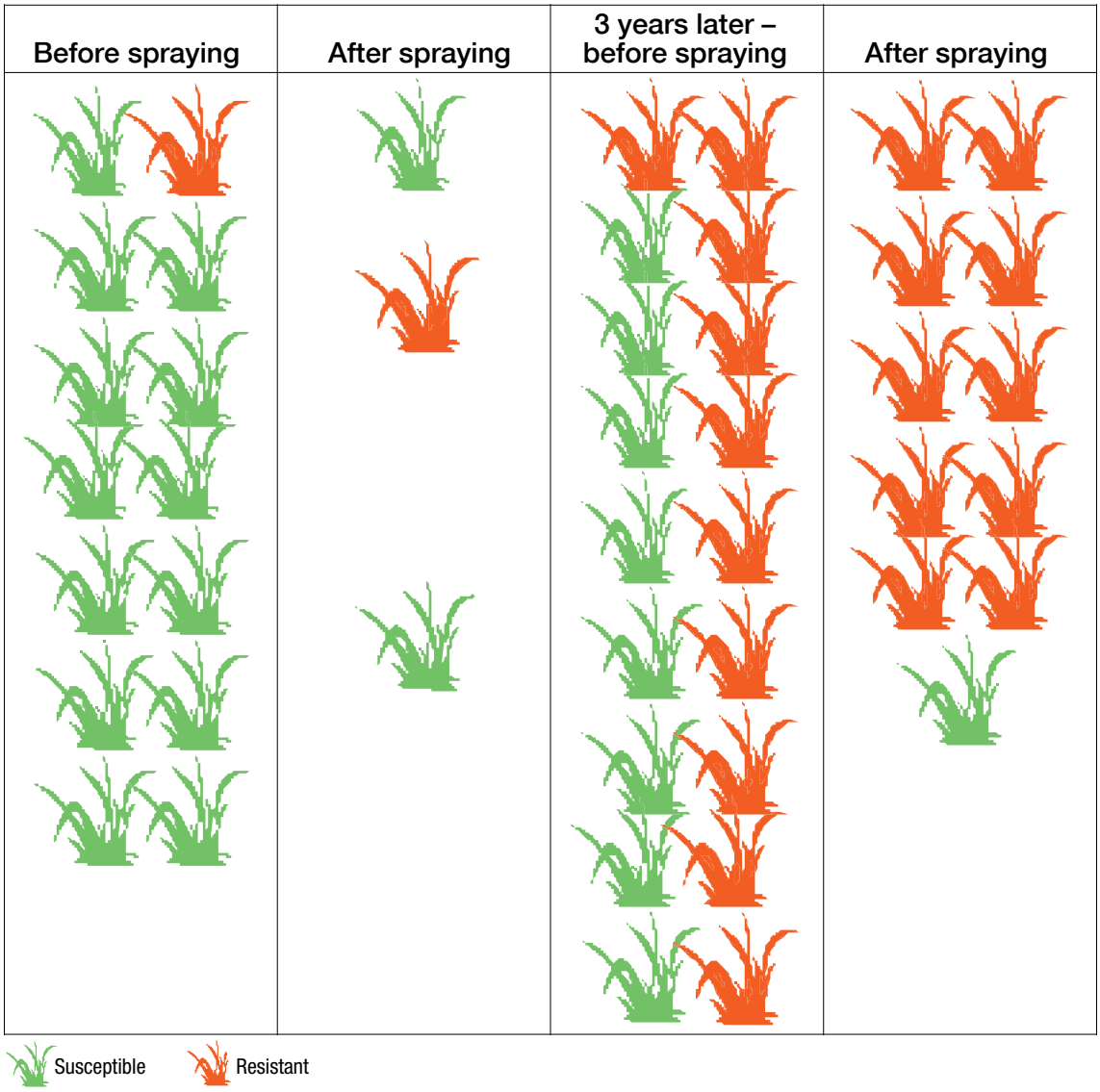
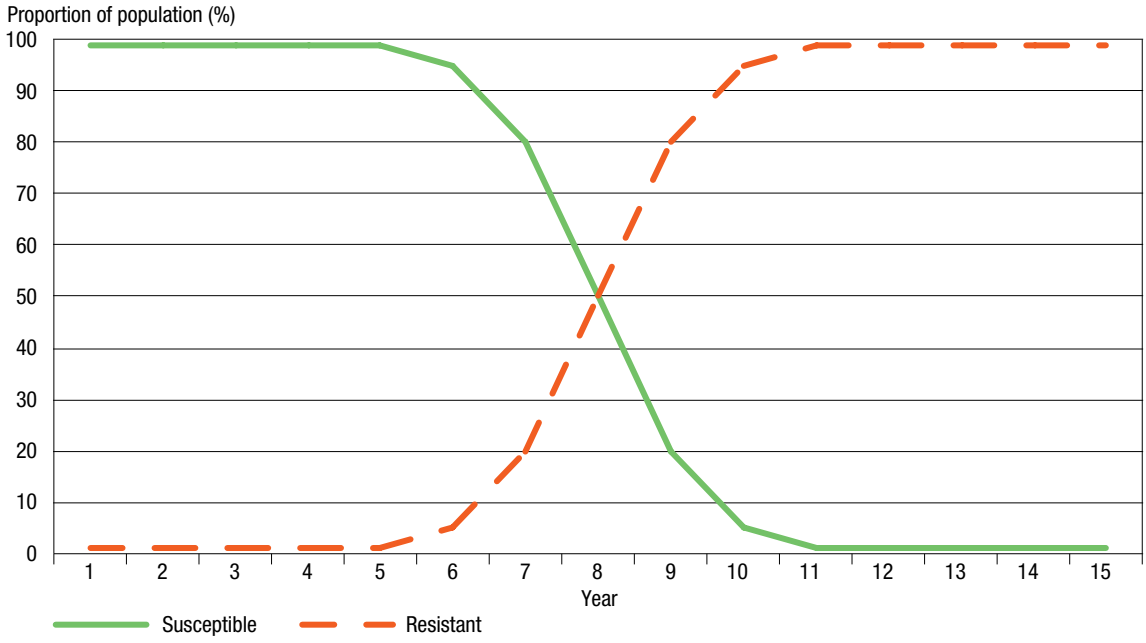


FIGURE HR2 **A generalised graph of the impact which repeated application of herbicides with the same MOA has on the proportion of susceptible and resistant plants.**



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, above).

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

2 **Introduction of resistance.** It is possible that resistance may not be present in the population initially, but is introduced as a weed seed contaminant in crop seed or fodder, on machinery or on/in animals. Alternatively resistance can develop through the arrival of wind- or water-driven resistant seeds or pollen. For example, species such as sowthistle (*Sonchus oleraceus*) and fleabane (*Conyza* spp.) can be spread up to 2 km by the wind. Pollen can also be dispersed great distances although the percentage able to successfully pollinate another plant at distances greater than 10 m is low. It has been found that grass pollen survives in the environment for up to three hours, with 1 per cent viable after two hours (Fei and Nelson 2003). However, Busi *et al* (2008) found that annual ryegrass pollen can fertilise plants up to 3 km distant when pollen competition from nearby plants is low. Flood water also has the potential to move a wide range of weed seeds over large distances.

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 will affect the number of herbicide applications before the general population becomes resistant to that herbicide.

These include:

- initial frequency of resistance gene(s) and MOA of the applied herbicide
- size of the weed population
- proportion of the weed population treated
- herbicide efficacy
- weed biological factors.

Initial frequency of resistance gene

The frequency of resistant individuals present in a population prior to herbicide application varies for different herbicide MOAs.

For example, high initial resistance in three untreated annual ryegrass populations (Table HR1, below) 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 8000
Group B	imazapyr	1 plant in 100,000 to 1 in 17,000

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

TABLE HR2 Initial frequency of individuals of annual ryegrass, estimated by modelling (Diggle and Neve 2001).

	Group A e.g. diclofop-methyl	Group B e.g. chlorsulfuron	Group M e.g. glyphosate
Estimated initial frequency	1 plant in 1,000,000	1 plant in 10,000	1 plant in 100,000,000

Neve *et al* (2003) simulated the evolution of glyphosate resistance in annual ryegrass. Using an initial resistance frequency of one 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.

This also influences the number of times a herbicide can be applied prior to a weed population developing resistance. Table HR3 (below) shows some rules of thumb regarding the number of years of effective herbicide application before resistance evolves, according to the MOA of the herbicide being used.

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
G	10	Medium
H	10	Medium
I	>20	Medium
K	>15	Medium
L	>15	Medium
M	>12	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 (Group B 'SU') 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 oats 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 (Mansooji *et al* 1992); see Table HR4 (page 35).
- Barley grass (*Hordeum* spp.) in South Australia has evolved resistance to paraquat (Group L) in no-tillage systems after approximately 15 application years (Alizadeh *et al* 1998).
- Broadleaf weeds such as charlock (*Sinapis arvensis*), Indian hedge mustard (*Sisymbrium orientale*) and common sowthistle have evolved resistance to 'SU' herbicides (Group B) after only two to four applications to weed populations in grain regions across Australia (Boutsalis and Powles 1995).
- Seventeen years of intense wheat–lupin rotation in the northern grain belt of Western Australia with wheat spraying pre-emergent triasulfuron (Group B) followed by 2,4-D (Group I) post-emergent every year and lupins sprayed with simazine/atrazine (Group C) followed by diflufenican (Group F) most years. No seedset management tactics were used. Wild radish (*Raphanus raphanistrum*) is now resistant to 2,4-D as well as Group B and F herbicides (Walsh *et al* 2003).

- 20 to 30 years of herbicide-reliant cropping have led to the development of three confirmed populations of glyphosate resistant wild radish in Western Australia's northern cropping region (Ashworth *et al* in press) and four populations of glyphosate resistant sowthistle (*Sonchus* spp.) in northern New South Wales (T. Cook pers. comm.).

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 south-east 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: 1981–1989

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

In this case study, heavy reliance on Group A ('fops') herbicide for annual grass control (eight applications in nine years) was typical practice. To kill weed (seedlings) in the target area (*Tactic Group 2,* section 4, page 113) was the primary focus for weed management, and failure to include tactics from alternative Tactic Groups to control survivors of the herbicide applications led to the development of herbicide resistance.

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

This study also shows that the overuse of a single MOA subgroup (in this case eight applications of Group A 'fops' in nine years) can lead to resistance in other MOA subgroups (in this case 'dims') that were never used in this paddock.

Triallate usually gives around 85 per cent control of wild oats if applied correctly. The additional MOA herbicides listed in Table HR4 (page 35) were applied to target other weed problems such as annual ryegrass, rather than as a management technique for the Group A resistance in wild oats. Trifluralin is not labelled for wild oats control (it gives 60 to 70 per cent control). The glyphosate application was used as a pasture spray-top in one season.

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

MOA group	Herbicide	Number of applications 1981–1989
A ('fop')	Diclofop-methyl	3
	Fluazifop-butyl	3
	Haloxyfop-ethoxyethyl	2
D	Trifluralin	3
J	Triallate	1
M	Glyphosate	1

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 per cent weed control increased the rate of resistance development to a greater extent than herbicides resulting in 80 per cent weed control.

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.

Using herbicides selects for resistance if survivors are allowed to set seed.

Use of sub-optimal herbicide rates will enable individuals carrying any possible resistance mechanisms or genes to survive – both strong and weak resistance mechanisms, along with some susceptible individuals. If herbicides are used at robust rates at the right growth stage and conditions, then there is high mortality and also individuals carrying weak resistance mechanisms or genes will not survive. Individuals carrying strong resistance mechanisms will survive.

When spraying herbicides a high level of weed control should be targeted to avoid loss of crop yield. A high level of control is determined by herbicide efficacy rather than by rate. For example, weed control in the order of 95 per cent may be obtained under optimal spraying conditions, while twice the recommended rate would be required to obtain the same level of control under poor spraying conditions or with poor application techniques.

ALWAYS USE ROBUST LABEL RATES OF HERBICIDE APPLIED TO MAXIMISE THEIR EFFICACY.

It is important to use a robust rate for maximum weed kill, but it is also necessary to kill survivors of the herbicide application using other tactics.

Weed population size

The larger the weed population, the greater the likelihood there will be of naturally occurring herbicide resistant individuals within the population.

A useful analogy in understanding the influence of weed population size is the presence of white-flowered individuals in a Paterson's curse (*Echium plantagineum*) 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.

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, such as the use of glyphosate

to control barnyard grass (*Echinochloa* spp.) occurring in summer fallows in the northern cropping region. It could also occur where a herbicide is applied late after more weeds have emerged (e.g. 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 on very susceptible species.

PHOTO: STEVE SUTHERLAND

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

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 (e.g. 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. Despite this, most fitness penalties incurred by herbicide resistance will be too small to have any effect on management within the paddock.

PHOTO: ANDREW STORRIE

Glyphosate-resistant awnless barnyard grass in grain sorghum, northern NSW.

PHOTO: ANDREW STORRIE

2,4-D resistant wild radish in a wheat crop, Wongan Hills, WA.

- **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^2 and wild radish and charlock around 30,000 seeds/m^2.
- **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 stockfeed, hay, crop seed, machinery and soil or animal movement. This is particularly important with forms that are naturally rare within a weed population such as glyphosate resistance.
- **Chance**
  The distribution of resistant individuals within a population is not uniform. On average, all ryegrass populations start off with about one plant in 17,000 with resistance to Group B herbicides. In reality, some populations have one plant in 8000, and others one 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) provides details of worldwide 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, page 38) and broadleaf (Table HR6, page 39) weed species have populations which have been confirmed to be resistant to a range of herbicides across Australia.

Extent of resistance to selective herbicides in Australia

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

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

TABLE HR5 Known populations of herbicide resistant grass weeds In Australia (updated by Storrie 2014).

Weed species	Herbicide group	Example herbicide	States with confirmed resistant populations					
			WA	SA	Vic	NSW	Tas	Qld
Annual ryegrass (*Lolium rigidum*)	A – 'fops'	Diclofop-methyl	X	X	X	X	X	
	A – 'dims'	Sethoxydim	X	X	X	X	X	
	B – sulfonylureas	Chlorsulfuron	X	X	X	X	X	
	B – imidazolinones	Imazapic, imazapyr	X	X	X	X	X	
	C – triazines	Simazine, atrazine	X	X	X			
	C – substituted ureas	Diuron		X	X			
	D – dinitroanilines	Trifluralin	X	X	X	X	X	
	L – bipyridiliums	Paraquat	X	X				
	M – glycines	Glyphosate	X	X	X	X		
	Q – triazoles	Amitrole	X	X				
Awnless barnyard grass (*Echinochloa colona*)	C – triazines	Atrazine	X			X		
	M – glycines	Glyphosate	X			X		X
Barley grass (*Hordeum leporinum*)	A – 'fops'	Haloxyfop, fluazifop		X	X	X	X	
	A – 'dims'	Sethoxydim		X				
	L – bipyridiliums	Paraquat	X	X	X	X	X	
	B – sulfonylureas	Sulfosulfuron/ Sulfometuron	X					
	B – imidazolinones	Imazamox, imazapic	X					
Barley grass (*Hordeum glaucum*)	A – 'fops'	Fluazifop		X				
	B – sulfonylureas	Sulfosulfuron/ Sulfometuron	X					
	L – bipyridiliums	Paraquat		X	X			
Brome grass (*Bromus diandrus*)	A – 'fops'	Haloxyfop			X			
	B – sulfonylureas	Mesosulfuron			X			
	B – sulfonamides	Pyroxulam			X			
	M – glycines	Glyphosate		X	X			
Brome grass (*Bromus rigidus*)	A – 'fops'	Quizalofop	X	X				
Red brome (*Bromus rubens*)	M - glycines	Glyphosate	X					
Giant Parramatta grass (*Sporobolus fertilis*)	J – alkanoic acids	Fluproponate				X		
Large crabgrass (*Digitaria sanguinalis*)	A - 'fops'	Fluazifop, haloxyfop	X	X				
	B - imidazolinones	Imazethapyr		X				
Liverseed grass (*Urochloa panicoides*)	C – triazines	Atrazine						X
	M – glycines	Glyphosate				X		
Paradoxa grass (*Phalaris paradoxa*)	A – 'fops'	Fluazifop				X		
	A – 'dims'	Sethoxydim				X		
Serrated tussock (*Nassella trichotoma*)	J – alkanoic acids	Fluproponate			X			
Silver grass (*Vulpia* spp.)	C – triazines	Simazine			X			
	L – bipyridilium	Paraquat			X			
Sweet summer grass (*Brachiaria eruciformis*)	M - glycines	Glyphosate						X
Annual veldt grass (*Ehrharta longifolia*)	A – 'fops'	Haloxyfop-r	X					
	A – 'dims'	Clethodim	X					
Wild oats (*Avena* spp.)	A – 'fops'	Diclofop-methyl	X	X	X	X		X
	A – 'dims'	Tralkoxydim	X	X	X	X		X
	B – sulfonylureas	Iodosulfuron-methyl-sodium		X		X		
		Flamprop-methyl						
	Z – aminopropionates			X	X	X		X
Windmill grass (*Chloris truncata*)	M – glycines	Glyphosate	X		X	X		
Winter grass (*Poa annua*)	Z – dicarboxylic acid	Endothal			X			

Note: Collated from information presented at www.weedscience.org/in.asp and other published literature.

TABLE HR6 Known populations of herbicide resistant broadleaf weeds in Australia (updated by Storrie 2014).

Weed species	Herbicide group	Example herbicide	States with confirmed resistant populations					
			WA	SA	Vic	NSW	Tas	Qld
African turnip weed (*Sisymbrium thellungi*)	B – sulfonylureas	Chlorsulfuron						X
Arrowhead (*Sagittaria montevidensis*)	B – sulfonylureas	Bensulfuron				X		
Black bindweed (*Fallopia convolvulus*)	B – sulfonylureas	Chlorsulfuron						X
Calomba daisy (*Pentzia suffruticosa*)	B – sulfonylureas	Metsulfuron-methyl		X				
Capeweed (*Arctotheca calendula*)	L – bipyridiliums	Paraquat, diquat			X			
Charlock (*Sinapis arvensis*)	B – sulfonylureas	Chlorsulfuron				X		
Common sowthistle (*Sonchus oleraceus*)	B – sulfonylureas M – glycines I – phenoxies	Chlorsulfuron Glyphosate 2, 4-D	X	X X	X	 X		X
Dense-flowered fumitory (*Fumaria densiflora*)	D – dinitroanilines	Trifluralin		X		X		
Flaxleaf fleabane (*Conyza bonariensis*)	M – glycines B – sulfonylureas	Glyphosate Chlorsulfuron		X X		X		X
Indian hedge mustard (*Sisymbrium orientale*)	B – sulfonylureas B – sulfonamides B – imidazolinones I – phenoxies	Chlorsulfuron Metosulam Imazethapyr 2,4-D	X X	X X X X	X	X X		X X
Paterson's curse / salvation Jane (*Echium plantagineum*)	B – sulfonylureas B – sulfonamides	Chlorsulfuron Metosulam	X X	X X				
Prickly lettuce (*Latuca serriola*)	B – sulfonylureas B – imidazolinones M - glycines	Chlorsulfuron Metosulam Glyphosate		X X	X X			
Sand rocket (*Diplotaxis tenuifolia*)	B – sulfonylureas	Chlorsulfuron		X				
Small square weed (*Mitracarpus hirtus*)	L – bipyridiliums	Paraquat						X
Starfruit (*Damasonium minus*)	B – sulfonylureas	Bensulfuron				X		
Stinging nettle (*Urtica urens*)	C – triazines	Simazine, atrazine			X			
Three-horned bedstraw (*Galium tricornutum*)	B – sulfonylureas B – imidazolinones B – sulfonamides	Sulfometuron Imazapyr Metosulam		X X X				
Turnip weed (*Rapistrum rugosum*)	B – sulfonylureas	Chlorsulfuron				X		X
Wild radish (*Raphanus raphanistrum*)	B – sulfonylureas B – sulfonamides B – imidazolinones C – triazines C – triazinones F – nicotinanalides I – phenoxies M – glycines	Chlorsulfuron Metosulam Imazapic, imazapyr Simazine, atrazine Metribuzin Diflufenican 2,4-D Glyphosate	X X X X X X X X	X X X X X	X X X	X		
Wild turnip (*Brassica tournefortii*)	B – sulfonylureas B – sulfonamides	Chlorsulfuron Metosulam	X	X X				

Note: Collated from information presented at www.weedscience.org/in.asp and other published literature.

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

The same survey revealed that 68 per cent of annual ryegrass populations were resistant to diclofop-methyl (Group A 'fop') and 88 per cent of populations were resistant to sulfometuron (Group B 'SU') which was a 20 per cent increase in the five years since the previous survey. Sixty-four per cent of these populations were resistant to both herbicide MOAs. Also 24 per cent of populations were developing resistance (1 to 20 per cent survival) to trifluralin (Group D) and 8 per cent to clethodim, a Group A 'dim' (Owen *et al* 2007).

In 2010 during another random survey conducted in Western Australia annual ryegrass seed was collected from 362 of the nearly 470 fields visited (Owen *et al* 2014). The results for the percentage of annual ryegrass populations and the increase in resistance since the 2003 survey are shown in Table HR7, below.

TABLE HR7 Increase in herbicide resistance in annual ryegrass in Western Australia since the 2003 survey (Owen *et al* 2014).

Herbicide	Percentage of populations with resistant plants	Percentage increase since 2003
Diclofop-methyl	96	28
Clethodim (250 mL/ha)	65	57
Clethodim (500 mL/ha)	42	–
Sulfometuron	98	10
Trifluralin	27	3
Atrazine	2	1
Glyphosate	7	6
Paraquat	0	–

Group A 'fops' and B herbicides are nearing 100 per cent resistant, while the increase in trifluralin resistance has been quite slow at 3 per cent.

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 similar surveys of annual ryegrass populations conducted in South Australia in 1998, 2003 and 2008 (Table HR8, below), the level of Group A resistance doubled, that of Group B resistance increased fourfold, and multiple resistance increased thirtyfold in five years (Preston 2004). Between 2003 and 2008 no further increases in resistance were observed.

TABLE HR8 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).

	Resistance in annual ryegrass populations in South Australia (%) by survey year		
	1998 (196 samples)	2003 (187 samples)	2008 (270 samples)
Group A herbicides	38	76	76
Group B herbicides	21	75	73
Group A and Group B herbicides	2	60	48

A survey of 270 annual ryegrass populations collected (Boutsalis *et al* 2012) at random in the mid-north and upper Yorke Peninsula of South Australia in 2008 found field-level resistance to Group D

herbicides in almost half of the populations (Table HR9, below). These populations were also found to have high levels of resistance to Group A and B herbicides (Boutsalis 2006, Boutsalis *et al* 2012).

TABLE HR9 Results of herbicide resistance in 270 randomly collected populations of annual ryegrass from the mid-north and upper Yorke Peninsula in 2008. Data show the percentage of samples with > 20% resistant individuals (measured as % survival) (Boutsalis *et al* 2012).

	MOA group A – 'fop'	MOA group A – 'dim'	MOA group B	MOA group D
Herbicide	Diclofop-methyl	Tralkoxydim	Chlorsulfuron	Trifluralin
Rate per ha	1500 mL	500 g	20 g	500 mL*
% survival	76	64	73	40

* Seed on the surface of pots is sprayed directly resulting in 100% coverage. This level of control is not generally achievable in the field and corresponds to rates in excess of 3 L/ha of trifluralin in low stubble situations.

Random surveys conducted in southern New South Wales in 1991 and 2007 (Table HR10, below) showed a significant increase in Group A and Group B resistance over this period (Broster *et al* 2011).

TABLE HR10 Number of herbicide resistant populations from two random surveys in southern New South Wales (Broster *et al* 2011).

Herbicide MOA	Herbicide	Year of survey	
		1991	2007
A – 'fop'	Diclofop methyl	14	81
A – 'dim'	Sethoxydim	12	43#
Group B – 'SU'	Chlorsulfuron	11	70
Group B – 'imi'	Imazapic/imazapyr	–-	65

Sethoxydim was only screened on diclofop-resistant populations in 2007.

A review of 10 years of herbicide resistance testing (1991 to 2001) conducted by Charles Sturt University found that the level of resistance in samples remained relatively constant across the years, although the number of postcode areas where samples originated increased by 18 per cent per year (Broster and Pratley 2006). This suggests that herbicide resistance was increasing in newer or previously less intensively cropped areas.

A random survey of paddocks in northern New South Wales and Queensland in 2003 identified approximately 10 per cent had wild oats 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 (*Rapistrum rugosum*), African turnip weed (*Sisymbrium thellungii*) and charlock. One paddock had Group C resistant barnyard grass (*Echinochloa* spp.) (Widderick and Galea 2004).

A survey in 2007 in the same area found 20 per cent of randomly tested wild oats populations had resistance to fenoxaprop and clodinafop. In the same project problem paddocks were also tested, with 85 per cent showing resistance and 60 per cent of these having resistance to three or more herbicides. Overall, 25 per cent of the samples that were resistant to fenoxaprop and clodinafop also had resistance to flamprop methyl which is in Group Z (Cook 2011).

This case study is from the northern region of the Western Australian wheatbelt renowned for large wild radish populations. As seen in Table HR11 (page 42), the high-risk Group B herbicides along with Group C 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. Herbicides were the sole weed control tactic in the rotation 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.

Herbicide resistance case study #2

Multiple resistance in wild radish

Weed: Two wild radish populations collected from the northern wheatbelt, Western Australia

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

Herbicide use history: 1983–1999

Resistance profile: Population 1 resistant to:

Group I phenoxies (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 phenoxies (2,4-D)

Group F nicotinanalides (diflufenican)

The two populations of wild radish in this case study are now resistant to three MOAs. Although the herbicide MOA was rotated, seedset 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.

TABLE HR11 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 1983–1999	
		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 (glycine)	Glyphosate	9	9

Extent of resistance to non-selective herbicides in Australia

Glyphosate

In 1996 glyphosate resistance was confirmed for the first time in annual ryegrass in Australia (Pratley *et al* 1996). It was documented in populations of awnless barnyard grass (*Echinochloa colona*) in New South Wales in 2007, and in 2008 glyphosate resistance occurred in populations of liverseed grass (*Urochloa panicoides*) also in New South Wales. In 2010 glyphosate resistance was documented in populations of flaxleaf fleabane (*Conyza bonariensis*) in Queensland and New South Wales and in windmill grass (*Chloris truncata*) in New South Wales. In 2011 brome grass (*Bromus diandrus*) was confirmed as resistant to glyphosate in South Australia. By mid 2014 glyphosate resistance was confirmed in wild radish (*Raphaus raphanistrum*), sowthistle (*Sonchus* spp.), red brome (*Bromus rubens*), sweet summer grass (*Brachiaria eruciformis*) and prickly lettuce (*Lactuca serriola*) (Preston 2014).

In May 2014 there were 574 documented glyphosate resistant populations of annual ryegrass, 98 of awnless barnyard grass, 58 of fleabane, 4 of sowthistle, 1 of prickly lettuce, 3 of wild radish, 11 of windmill grass, 4 of liverseed grass, 5 of great brome and 1 of red brome.

PHOTO: ANDREW STORRIE

Field heavily infested with glyphosate resistant annual ryegrass having been sprayed with 2 L glyphosate 450 per ha.

As with all other herbicides at risk of evolving resistant weed populations, selection for resistance to glyphosate is promoted by particular management activities (Table HR12, below). It is important to avoid 'risk-increasing' actions and include 'risk-decreasing' tactics.

TABLE HR12 Factors that influence the risk of the evolution of resistance to glyphosate (Australian Glyphosate Sustainability Working Group 2014).

Risk-increasing actions	Risk-decreasing actions
• continual reliance on glyphosate before seeding • lack of tillage • lack of effective in-crop weed control • frequent glyphosate-based chemical fallow • inter-row glyphosate use (unregistered) • frequent late season weed control and in-crop spray-topping with glyphosate • over-reliance on glyphosate resistant crops • high weed numbers	• non-herbicide practices to prevent formation of viable weed seed • using crops with high levels of competition with weeds • using late season weed control and in-crop spray-topping with alternative herbicide groups • farm hygiene to prevent movement of resistant seed • the double knock technique* • strategic use of alternative knockdown groups • use of alternative herbicide groups or tillage for inter-row and fallow weed control • effective in-crop weed control • full-disturbance cultivation at sowing • applying stewardship plans when growing glyphosate resistant crops

* A double knock tactic is where the second treatment controls the survivors of the first treatment. This includes two consecutive herbicide applications with different modes-of-action, or could be a herbicide followed by cultivation or heavy grazing.

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 practices 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 then 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: 1981–1999

Resistance profile: The high number of glyphosate applications (Table HR13, page 44) on this population resulted in the evolution of glyphosate resistance; the population was not resistant to Group A or B herbicides.

This herbicide use profile is typical 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 by the grower in this case study. Introduction of no-till systems reduced crop competition by going to wider row spacings (from 18 cm to 38 cm for wheat). Post-emergent grass herbicides were targeted at wild oats and not annual ryegrass.

During the 1990s the problem of rising watertables and the threat of atrazine (Group C) contamination of groundwater were highlighted to growers. The concept of 'opportunity cropping' (sowing the most suitable crop when the soil profile contains 1 m of 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. Growers maintained the ability to 'opportunity crop' by ceasing to use residual herbicides; however, fallows of six to 18 months' duration remained the norm. 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.

No tactics to deplete weed seed in the target area soil seedbank (*Tactic Group 1,* section 4, page 92), stop weed seedset (*Tactic Group 3,* section 4, page 170), or to prevent viable weed seeds within the target area being added to the soil seedbank (*Tactic Group 4,* section 4, page 212) were used. Growers and agronomists were not monitoring levels of weed control following spraying or changes in weed species and number, so no risk-reducing measures were taken until resistance was suspected.

TABLE HR13 Number of herbicide applications for New South Wales herbicide resistance case study #3 (Storrie and Cook 2002).

MOA group	Herbicide	Number of applications 1981–1999
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

Paraquat

Three populations of paraquat resistant annual ryegrass have been confirmed in south-eastern South Australia in 2010 by glasshouse experiments. One population was also resistant to glyphosate. Glyphosate resistance evolved on an irrigation channel and subsequently moved into the paddock, where it was then selected with paraquat. In September 2013 an annual ryegrass population from a Western Australian vineyard was confirmed resistant to both glyphosate and paraquat following a history of using both herbicides.

Other species have previously developed resistance to Group L herbicides in Australia, the first case being northern barley grass (*Hordeum glaucum*) in 1983 (Table HR14, page 45). Small square weed (*Mitracarpus hirtus*) was the first case of resistance to paraquat in Australia that developed outside broadacre agriculture.

All cases of resistance to paraquat are in situations with long histories of use (more than15 years).

TABLE HR14 Species that have developed resistance to paraquat in Australia.

Species	Common name	Year confirmed	State	Crop	Resistance to other MOAs / herbicides
Arctotheca calendula	Capeweed	1984	Victoria	Lucerne	diquat (L)
Hordeum glaucum	Northern barley grass	1983	Victoria	Lucerne	diquat (L)
Hordeum leporinum	Barley grass	1988	Victoria	Lucerne	diquat (L)
Lolium rigidum	Annual ryegrass	2010	South Australia	Pasture seed	Group A
Lolium rigidum	Annual ryegrass	2010	South Australia	Pasture seed	Group M
Lolium rigidum	Annual ryegrass	2013	Western Australia	Vineyard	Group M
Mitracarpus hirtus	Small square weed	2007	Queensland	Mangoes	diquat (L)
Vulpia bromoides	Silver grass	1990	Victoria	Lucerne	diquat (L)

Further information

Australian Glyphosate Sustainability Working Group

The Australian 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.

Its priority goals are to:

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.

The Australian Glyphosate Sustainability Working Group's website is supported by the Grains Research and Development Corporation, and key research- and development-based crop protection companies with an interest in the sustainability of glyphosate (www.glyphosateresistance.org.au). It 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 weed populations developing resistance to glyphosate (Table HR12, page 43) which are available as industry-specific posters on the website).

There is also an active register, containing information about all the known weed populations resistant to glyphosate and paraquat 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) 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 Integrated Weed Management plan, incorporating tactics from a number of Tactic Groups (see *Section 4 Tactics*, page 91) and following the recommendations listed in the Resistance Management Strategies, can extend the effective life of herbicides in crop paddocks and assist management of herbicide resistant weed populations.

TABLE HR15 Resistance watch: confirmed resistance in overseas populations of common weed species in crops (updated by Storrie 2014).

Weed species	Herbicide group	Example herbicide	Countries with confirmed resistant populations
Amaranthus blitum	B C L	Imazethapyr Triazines Paraquat	USA France Malaysia
Ball mustard (*Neslia paniculata*)	B – sulfonylureas	Metsulfuron-methyl	Canada
Barnyard grass (*Echinochloa* spp.)	A – 'fops' B – imidazolinones C – amides D – dinitroanilines J – thiocarbamates	Fenoxaprop, quizalofop Imazethapyr Propanil Pendimethalin Molinate	Thailand, USA Yugoslavia USA, Greece, Italy, Thailand Bulgaria USA, China
Brome grass (*Bromus* spp.)	C – triazines C – substituted ureas	Atrazine Chlorotoluron	France, Spain Spain
Charlock (*Sinapis arvensis*)	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 (*Stellaria media*)	B – sulfonylureas C – triazines I – phenoxies	Chlorsulfuron Atrazine Mecoprop	Canada, Denmark, Ireland, New Zealand, Norway, South Africa, Sweden, United Kingdom Germany United Kingdom
Crowsfoot grass (*Eleusine indica*)	A – 'fops' B – imidazolinones D – dinitroanalines L – bipyridiliums M – glycines	Fluazifop Imazapyr Trifluralin Paraquat Glyphosate	Brazil, Malaysia Costa Rica USA, Malaysia, USA Malaysia
Fleabane (*Conyza* spp.)	B – sulfonylureas C – triazines C – substituted ureas L – bipyridiliums	Chlorsulfuron Atrazine Linuron Paraquat	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
Green amaranth (*A. viridus*)	B + C	Trifloxysulfuron + triazines	Brazil
Lesser canary grass (*Phalaris minor*)	A – 'fops' B – sulfonylureas C – substituted ureas	Fluazifop Triasulfuron Isoproturon	Mexico, Israel, USA, South Africa South Africa India
Needle burr (*A. spinosus*)	M	Glyphosate	USA
Paradoxa grass (*Phalaris paradoxa*)	C – triazines	Atrazine	Israel
Powell's amaranth (*A. powellii*)	B C	Imazethapyr Triazines, ureas, amides	Canada, USA Canada, Europe, USA
Redroot amaranth (*A. retroflexus*)	B C	Imazethapyr Triazines, ureas, amides	Europe, USA Europe, USA
Shepherd's purse (*Capsella bursa-pastoris*)	C – triazines	Atrazine	Poland
Slim amaranth (*A. hybridus*)	B C	Imazethapyr Triazines, nitriles	USA Europe, South Africa, USA
Summer grass (*Digitaria sanguinalis*)	C – triazines	Atrazine	France, Poland
Summer grass (*Digitaria ciliaris*)	A – 'fops'	Fluazifop-p	Brazil
Wild oats (*Avena* spp.)	D – benzamides J – thiocarbamates	Propyzamide Tri-allates	USA, Canada USA
Winter grass (*Poa annua*)	B C D J L M Q	Trifloxysulfuron Simazine, prometryne, terbutryne Trifluralin Ethofumesate Paraquat Glyphosate Amitrole	USA, Europe, Japan, USA USA USA Europe USA Europe
Wireweed (*Polygonum aviculare*)	atrazine amitrole	Atrazine Amitrole	Belgium, Netherlands Belgium

Note: Collated from information presented at www.weedscience.org/in.asp and other published literature.

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 (Table HR5, page 38 and Table HR6, page 39). It is also important to know which weeds are likely to develop resistance.This will depend on biological characteristics of the plant and the farming system in which it grows.

Global examples of herbicide resistance are presented in Table HR15 (page 46). Although these weeds are present in Australia, to date no populations of the herbicide resistance shown in this table have been reported here in these species.

It is mostly winter weeds that are at greatest risk in southern and western cropping zones of Australia, 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).

A large number of weed species are present in the cropping region of north-eastern Australia, which includes northern New South Wales, southern Queensland and central Queensland, where a survey identified 105 weeds from 95 genera, with the major weeds being sowthistle, turnip weed, barnyard grass and liverseed grass (Osten *et al* 2007).

With this large number of weeds occurring in diverse farming systems it was seen as important to rank weeds species and farming systems at risk of developing glyphosate resistance (Thornby *et al* 2010; Thornby *et al* 2011; Werth *et al* 2011). The top 20 weeds in the north-east grain region are shown in Table HR16 (below), while the highest risk farming systems were summer fallow and both glyphosate resistant and non-glyphosate resistant, non-irrigated cotton. It is interesting to note that five species on the list have already developed glyphosate resistance in this region. This research has also shown that growers should identify their high risk weeds and rotations and tailor their management strategies around these rather than their most prevalent weeds.

TABLE HR16 Top 20 species in the north-eastern grain region at risk of developing glyphosate resistance (Werth *et al* 2011).

Species	Common name
Brachiaria eruciformis	Sweet summer grass
Conyza bonariensis	Flaxleaf fleabane
Urochloa panicoides	Liverseed grass
Chloris virgata	Feathertop Rhodes grass
Sonchus oleraceus	Sowthistle
Echinochloa colona	Awnless barnyard grass
Eleusine indica	Crowsfoot grass
Phalaris paradoxa	Paradoxa grass
Hordeum spp.	Barley grass
Lolium rigidum	Annual ryegrass
Dactyloctenium radulans	Button grass
Digitaria ciliaris	Summer grass
Chloris truncata	Windmill grass
Amaranthus hybridus	Redshank
Cirsium vulgare	Spear thistle
Silybum marianum	Variegated thistle
Sorghum halepense	Johnson grass
Eragrostis cilianensis	Stink grass
Avena spp.	Wild oats
Lactuca serriola	Prickly lettuce

The development of glyphosate resistance in annual ryegrass, awnless barnyard grass and liverseed grass will see the risks for Group A and Group L resistance increase in these species.

The risk for winter weeds is mainly the expansion of current known problems such as glyphosate resistance in annual ryegrass and wild oats, Group B resistance in brassica weeds and Group A and Z resistance in wild oats.

The extensive use of trifluralin (Group D) in no-till farming systems in southern Australia is a continuing high risk for resistance in annual ryegrass. Shepherd's purse (*Capsella bursa-pastoris*) is also at risk of developing Group B resistance.

Herbicide resistance testing

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

If done properly, herbicide resistance testing will tell the adviser and grower which herbicides are still effective on the target weeds in certain paddocks. This can save the unnecessary use of ineffective herbicides that are unable to kill the weeds in question; it will also optimise crop yield and provide essential information on in-crop and future weed management.

PHOTO: PETER BOUTSALIS

Foreground (left) trifluralin susceptible, (centre) trifluralin resistant biotype with intermediate resistance and (right) trifluralin resistant biotype with strong resistance. The array of pots in the background represent randomly collected 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.

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

Commercial testing services grow the plants under glasshouse conditions, removing any climatic or paddock variability that may affect the results, as well as using laboratory quality spraying equipment. They are able to easily test a number of different herbicides at several rates and compare the results to standard susceptible and resistant biotypes sprayed at the same time.

For information on how to test for resistance, see *Implementing an IWM program using tactic groups*, section 5, page 237 and the Australian Glyphosate Sustainability Working Group website (www.glyphosateresistance.org.au).

Contributors

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