

## Management of herbicide-resistant grass weeds in Europe

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### Summary

European agricultural methods, based mainly on herbicide weed control, have led to the appearance of 15 herbicide-resistant grass weed biotypes during the period from 1975 to 1995. Target site mutations and enhanced detoxification processes have been characterized as the main mechanisms of resistance. These biotypes are resistant to herbicide families such as PSI and PSII inhibitors, ACCase inhibitors, ALS inhibitors and auxin analogues, some resistant biotypes showing cross- and multiple-resistance. General guidelines to identify and control weed resistance in grass weeds are given. Specific herbicidal control methods which have succeeded in eradicating or at least controlling resistance are suggested for resistant biotypes with mutated target sites. Integrated weed management, including both cultural and chemical methods, is suggested in those cross-resistant biotypes showing enhanced detoxification as a mechanism of tolerance.

### Introduction

Traditional agricultural methods, based on chemical weed control, have increased the number of herbicide-resistant weeds and the area they infest in Europe at an alarming rate. In Europe, 15 herbicide-resistant grass weeds biotypes were discovered during the period from 1975 to 1995 (De Prado, 1995). Two mechanisms of resistance were mainly responsible for this tolerance to herbicides: target site mutations and the development of enhanced detoxification. Regarding the characteristics of the resistant weed biotypes, the terms used in this review are based on those suggested by Jutsum and Graham (1995): "Resistance" will be used where weed populations have acquired an inheritable ability to survive herbicide treatments that would, under normal conditions of use, effectively control that weed population. "Cross-resistance" describes plants with conferred resistance to several herbicides due to the presence of a single resistance mechanism. "Multiple resistance" will be used for weed populations which possess two or more distinct resistance mechanisms. The aim of this paper is to review the present situation regarding the evolution of herbicide-resistant grass weeds and their management in Europe.

### Grass weed management using herbicidal control methods

It has been widely accepted that it is easier to prevent or delay the risk of selecting resistant weed biotypes than to eradicate or control these biotypes after they have been selected by herbicide(s) and they are infesting a large area. Europe presents a huge diversity of cultural methods, each one fitted to the special agronomic characteristics of each country. Therefore, it is easier to change herbicide control strategies than to change mechanical methods used by farmers over many decades. Successful management of resistant weeds, and the range of alternative herbicides, depends upon the resistance spectrum or the extent of cross- and multiple resistance of these biotypes. Although it is not easy to develop a common program for

weed control, we can give some general rules which may help to minimize this problem.

#### Resistance to PSII inhibiting herbicides.

The most important problem related to herbicide resistance in Europe concerns weed biotypes resistant to the PSII-inhibiting herbicides. These biotypes have been mainly detected in France, Spain and the United Kingdom, infesting crops such as corn, wheat, rice, olive trees and orchards (Table 1).

Table 1. Grass weeds resistant to photosystem II-inhibiting herbicides

Species	Selection system	Crop	Location	Resistance Index	Mechanism of resistance
<i>Apera spica-venti</i>	Ch	Wh	Sw	--	D
<i>Alopecurus myosuroides</i>	Ch	Wh	Ge,UK,Sp	9-29.0 Ch	D
<i>Bromus tectorum</i>	At/Si	Co/Ol	Fr,Sp	73.0 Si	TS
<i>B. tectorum</i>	Ch	Wh	Sp	3.4 Ch	D
<i>Digitaria sanguinalis</i>	At	Co	Bu,Fr,Po	>5.0 Si	TS
<i>Echinochloa crus-galli</i>	At	Co	Au,Bu,Fr,Po, Sp	80.0 At	TS
<i>E. crus-galli</i>	Pr	Ri	Gr	>2.1 Pr	D
<i>Lolium rigidum</i>	Di	Wh	Sp	6.6 Ch	D
<i>L. rigidum</i>	Di+Ch	Wh	Sp	9.3 Ch	D
<i>L. rigidum</i>	Si	Ol	Sp	>10.0 At	TS
<i>Poa annua</i>	At	Co/Or	Be,Fr,Ge,Ne, UK,Sw	>6.0 At	TS
<i>Setaria faberi</i>	At	Co	Sp	10.0 At	TS
<i>S. glauca</i>	At	Co	Fr,Sp	12.1 At	TS
<i>S. viridis</i>	At	Co	Fr,Sp	6.5 At	TS

Selection System: At (atrazine); Ch (chlorotoluron); Di (diclofop-methyl); Pr (propanil); Si (simazine).

Crop: Co (corn); Ol (olive trees); Or (orchards); Ri (rice); Wh (wheat).

Location: Au (Austria); Be (Belgium); Bu (Bulgaria); Fr (France); Ge (Germany); Gr (Greece); Ne (The Netherlands); Po (Poland); Sp (Spain); Sw (Switzerland); UK (United Kingdom).

Resistance Index: ED<sub>50</sub> resistant biotype/ED<sub>50</sub> susceptible biotype.

Mechanism of resistance: D (enhanced herbicide detoxification); TS (mutated target site).

#### Grass weed biotypes resistant to s-triazines.

The continuous use of s-triazines for more than 10 years has led to the emergence of seven s-triazine-resistant weed biotypes in atrazine plus cyanazine-treated corn fields and two s-triazine-resistant weed biotypes in simazine-treated olive tree fields (Table 1). These biotypes are cross-resistant to other triazine herbicides at the chloroplastic level as has been demonstrated for *Setaria* spp and *Bromus tectorum* (Romera and De Prado, unpublished data).

The most effective way to control these triazine-resistant biotypes in corn is to rotate herbicides or mixture of herbicides such as sulfonyleureas and chloroacetamides+triazines. Other weed control programs could be the use of vigorous, high-quality seeds which compete with the weed and/or herbicide-resistant crops (Shaner *et al.*, 1996; Somers, 1996).

In olive tree fields, where non-tillage methods reduce soil erosion and improve water balances in the drought-stricken fields in comparison with conventional tillage, two simazine-resistant biotypes (*B. tectorum* and *L. rigidum*) have been detected. In such cases methods such as *spray topping*, using non-selective herbicides (glyphosate, paraquat+diquat) at a late stage of weed growth, have resulted in preventing seed production and subsequent eradication (Powles *et al.*, 1992).

#### *Grass weed biotypes resistant to substituted ureas*

Several chlorotoluron-resistant biotypes of *Apera spica-venti*, *Alopecurus myosuroides*, *Bromus tectorum* and *Lolium rigidum* have been reported in European winter wheat fields continuously treated with substituted ureas or/and ACCase-inhibiting herbicides (Table 1). Although the mechanism of resistance in *A. spica-venti* is still unknown, the other three species showed enhanced chlorotoluron metabolism (Kemp *et al.*, 1990; Jorin *et al.*, 1992; Menendez *et al.*, 1995a; De Prado *et al.*, 1995). Cross-resistance conferred by enhanced detoxification cannot be controlled only by herbicides. Alternative control methods such as Integrated Weed Management (IWM) programs are needed. Applied to herbicide resistance management, IWM would consist of (A) identifying the critical periods of life cycle of herbicide-resistant biotypes when they are more susceptible to be controlled, and (B) employing both cultural and rational chemical methods during those periods to eradicate these biotypes.

#### *Grass weed biotypes resistant to propanil*

Barnyardgrass (*Echinochloa crus-galli*) is one of the most troublesome weeds in rice, causing serious yield losses. Postemergence use of propanil at rates of 1 to 4 Kg a.i. ha<sup>-1</sup> provides satisfactory control of the weed. However, in 1986 a propanil-resistant biotype of *E. crus-galli* appeared in Greece, where rice had been grown for over a decade and propanil was used repeatedly (Giannopolitis and Vassiliou, 1990). Enhanced metabolism of propanil is the operative mechanism of resistance. Management of *E. crus-galli* has been successfully achieved using alternative herbicides (molinate and quinclorac) or mixtures and sequences of herbicides (molinate+bensulfuron, molinate+thiobencarb). Other strategies could be the use of synergists and safeners (Hatzios, 1991; Lamoureux and Rusness, 1994) and, in the future, the use of glyphosate-resistant rice (Fisher, 1995, personal communication).

#### *Resistance to PSI-disrupting herbicides.*

Resistance to paraquat has been identified in Europe in two grass species, *Poa annua* and *Lolium perenne* (Clay, 1989; Faulkner, 1976). The *Poa annua* biotypes were harvested in 1988 in England from two hops gardens treated annually with paraquat and simazine for about 25 years. These biotypes were resistant to both herbicides, although the level of resistance to simazine was slight. Paraquat-resistant *L. perenne* was selected by the Northern Ireland Plant Breeding Station. Management of these biotypes could be successfully achieved by using herbicide rotations with different mode of action to paraquat and/or to triazine herbicides.

#### *Resistance to ACCase-inhibiting herbicides*

Resistance to acetyl coenzyme-A carboxylase-inhibiting herbicides was first confirmed in England (Moss, 1990) and Spain (Menendez *et al.*, 1993), where different *Alopecurus myosuroides* populations proved resistant to chlorotoluron and cross-resistant to some graminicides. Since 1990, other diclofop-methyl-resistant biotypes of four different species have been found in Spain, France, Switzerland and England (Table 2), showing cross- and multiple-resistance to other herbicide families (Mayor and Maillard, 1995; Moss, 1995; Gasquez *et al.*, 1995; Menendez *et al.*, 1995b). These biotypes showed at least one of these three mechanisms of resistance to diclofop-methyl: (A) an altered ACCase target site, (B) enhanced herbicide metabolism and (C) recovery of plasma membrane potential.

#### *Resistance due to an altered ACCase target site.*

Three diclofop-methyl-resistant biotypes of *Lolium rigidum* (Spain), *L. multiflorum* (France) and *Avena fatua* (England) have been reported recently (Table 2). In these biotypes tolerance to graminicides (aryloxyphenoxypropanoates [AOPP] and cyclohexanediones or [CHD]) was due to a mutated form of ACCase. These three diclofop-resistant biotypes are only cross-resistant to other AOPPs and CHDs, but they were more resistant to the former than to the latter. In contrast, they were susceptible to other herbicides with different target sites such as ALS inhibitors, PSI and PSII inhibitors and tubulin formation inhibitors..

Table 2. Grass weeds resistant to ACCase-inhibiting herbicides.

Species	Selection system	Crop	Location	ED50 R (kg a.i. ha <sup>-1</sup> )	Mechanism of resistance
<i>Avena fatua</i>	Di	Wh	UK	-	TS
<i>Apera spica-venti</i>	Ch	Wh	Sw	-	nd
<i>Alopecurus myosuroides</i>	Ch	Wh	UK,Sp	7.5-3.9 Di	D+RMP
<i>A. myosuroides</i>	Cl+Di	Wh	UK	13.7 Di	nd
<i>Lolium multiflorum</i>	Di	Wh	Fr	>10.0 Di	TS+RMP
<i>L. rigidum</i>	Ch+Di	Wh	Sp	>1.5 Di	D+RMP
<i>L. rigidum</i>	Di	Wh	Sp	>1.5 Di	RMP
<i>L. rigidum</i>	Di	Wh	Sp	>10.0 Di	TS+RMP

Selection System: Ch (chlorotoluron); Cl (clodinafop-propargyl); Di (diclofop-methyl).

Mechanism of resistance: D (enhanced herbicide detoxification); RMP (recovery of membrane potential), TS (mutated target site), nd (not determined).

Diclofop-resistant wild oat has been successfully controlled on the Canadian prairies by using crop and herbicide rotation. This herbicide rotation includes triallate alone or formulated with trifluralin in cereals, and trifluralin in oil seed crops (Morrison and Bourgeois, 1995). However, the identification of multiple-resistance in some Canadian *A. fatua* and European *L. rigidum* biotypes requires the adoption of IWM programs. These methods should include the use of cleaned equipment before harvesting, especially when moving to different fields, vigorous crop growth to outcompete resistant weeds, growing highly competitive crops such as winter wheat, and using herbicide-resistant wheat (Morrison and Bourgeois, 1995; Shaner *et al.*, 1996).

#### Resistance due to enhanced herbicide metabolism

Resistance to diclofop-methyl has been detected in three biotypes of *Alopecurus myosuroides*, *Lolium rigidum* and *Apera spica-venti* harvested from winter wheat field treated with chlorotoluron as main herbicide (Table 2). The two former biotypes showed an ability to detoxify diclofop-methyl (Menendez *et al.*, 1993; Menendez *et al.*, 1995b). The mechanism of resistance in the *A. spica-venti* biotype is still unknown. However, the presence of cross-resistance in this weed biotype could point to enhanced metabolism as its main mechanism of resistance, as has been demonstrated for *A. myosuroides* and *L. rigidum*. Herbicidal control is not easy under these circumstances. Management studies carried out on *A. myosuroides* in the UK using pre-emergence treatments of triallate followed by clodinafop-propargyl with or without trifluralin and annually alternating mixtures of clodinafop-propargyl with either trifluralin or isoproturon gave the most promising results (Corners *et al.*, 1995). However, glasshouse studies carried out with *A. myosuroides* and *L. rigidum* have shown that these biotypes showed cross-resistance to clodinafop-propargyl, isoproturon and triallate. In such cases, non-selective herbicides such as glyphosate have succeeded in controlling these biotypes at much lower rates than those used in normal agriculture practices (Menendez and De Prado, 1995).

#### Resistance by recovery of plasma membrane potential.

Recovery of membrane polarity has been detected in Europe in aryloxyphenoxypropionate-resistant biotypes of *A. myosuroides*, *L. rigidum* and *L. multiflorum* alone or together with one of the mechanisms previously described, as a multiple-resistance phenomenon. European biotypes showing polarity recovery display three different resistance patterns (De Prado, unpublished data) (Table 2). *A. myosuroides* exhibited polarity recovery plus enhanced detoxification, while *L. multiflorum* showed polarity recovery plus a mutated ACCase form. Regarding diclofop-methyl-resistant *L. rigidum* biotypes, these can be included in both groups, with a new biotype characterized as diclofop-resistant due only to membrane polarity recovery, but which shows multiple-resistance to several herbicide families by enhanced detoxification.

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