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Weed Resistance to Herbicides–Mechanisms and Molecular Basis

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Abstract: Herbicide resistance is increasing throughout the world in a rapid and threatening pace. Resistance is usually associated with high selection pressure imposed on the weed population by using high rates of residual herbicides, monoculture and minimum tillage. As an answer to such a strong selection impact and due to the fact that one herbicide or those having the same mechanism of impact are successively used, the genetic composition of weed populations changes in that the frequency of resistant alleles and individuals increases. Re-use of the same herbicide or of that with the same impact mode disrupts the sensitive and makes the resistant populations viable so that the characteristics of resistance may further be transferred to the next generation.

This review summarises information from the literature and experimental experience of the authors in research on weed resistance to herbicides. Factors conditioning the origin of resistance are described. The origin of resistant weeds to nine active ingredients with a different mode of action is presented chronologically and the distribution of resistant weeds around the world outlined. The fundamental modes of action: reduction of the target site sensitivity, so-called „target site resistance“, and the mode by which a herbicide is metabolised into inactive products, are listed. Function and genetic modifications of target sites of selected herbicides are described.

Key words: resistance to herbicide, mechanisms and molecular basis of resistance.

Introduction

Resistance to different herbicide classes has appeared in numerous plant species throughout the world, particularly if and where monoculture, monoherbicide and minimum tillage are widely practiced (Rubin 1991). The repetitive use of high rates of the same residual herbicide (mostly triazines) along roadsides and railways, where cultivation is impractical or impossible, also led to the appearance of herbicide resistant populations (Rubin et al., 1985; Chodová and Mikulka 2000; Salava et al., 2004). The application of the same herbicide with long-lasting soil activity every season and repetitive application of a postemergence herbicide several times a year may result in an increase of selection pressure and lead to resistance.

Resistance is not due to mutation caused by a herbicide, rather it arises from the selection of a spontaneous mutation or of small pre-existing populations of resistant plants through the selection pressure exerted by the herbicide (Duke et al., 1991). The rapid spread of herbicide resistance endangers the usefulness of valuable old and new herbicide classes. It also increases the cost of weed control and poses a real threat to the environment (Rubin 1991).

LeBaron and Gressel (1982) used the term resistance as defined by the FAO "Herbicide resistance is the inherent activity of a species to survive and reproduce following exposure to a dose of herbicide normally lethal to its wild type". According to Rubin (1991) the term "Resistance will be used where a population has acquired an inheritable capacity to withstand herbicides applied at or above field rate".

Herbicide resistance has appeared not only when residual herbicides were heavily used, but also in cases where the selection pressure is inflicted by the repeated use of herbicides like paraquat which lack soil activity (Fuerst and Vaughn 1990; Rubin 1991). New cases of a weed being resistant to different herbicides have already appeared.

Resistance Risk Assessment

Herbicides such as triazines, phenylureas, aryloxyphenoxypropanoates, cyclohexanodiones, sulfonyleureas and imidazolinones have often been used continuously without any rotation of crops and herbicides (De Prado et al., 1997). The poor agricultural practice of monocultures has led to soil and environmental problems, uncontrolled insects and disease, and herbicide resistance (Gressel 1997). Until now, 291 resistant biotypes belonging to 174 species (104 broadleaf and 70 grass weeds) have been identified as being resistant to various herbicides (Heap 2004). The number of herbicide resistant weed species differs between the areas (Tab. 1.).

Tab. 1. The number of weed species resistant to different herbicides (acetyl – CoA carboxylase inhibitors, acetolactate synthase inhibitors, triazines, ureas and amides, bipyridiliums and dinitroanilines) in some countries

Country	Total	ACCCase inhibitors	ALS inhibitors	Triazines	Ureas/amides	Bipyridiliums	Dinitro-anilines	Other
USA	107	15	37	20	7	3	6	19
Canada	43	2	17	12	3	2	1	6
Australia	42	8	16	4	0	4	2	8
France	30	5	1	22	1	0	0	1
Spain	26	1	3	18	3	0	0	1
United Kingdom	24	4	4	8	2	2	1	3
Israel	20	2	6	11	1	0	0	0
Germany	18	1	1	13	3	0	0	0
Belgium	18	2	1	7	1	3	1	3
Switzerland	14	0	0	11	3	0	0	0
Czech Republic	13	0	1	12	0	0	0	0

It is difficult to state the real number of herbicide resistant weed populations, as it is highly dependent on the intensity of the search (Van Oorschot 1991).

The problem was so serious that scientists have formed the Herbicide Resistance Action Committee (Hrac 2004) to standardise herbicide classification according to the mode of action, and to highlight management strategies for the control of resistant weeds. Most of the weeds resistant to herbicide belong to the botanical families *Poaceae*, *Amaranthaceae*, *Asteraceae*, *Polygonaceae* and *Chenopodiaceae* (De Prado et al., 1997).

Present State and Distribution of Herbicide Resistance

Weeds Resistant to Photosystem II inhibitors

Among the inhibitors of Photosystem II (PS II) are active ingredients from the chemical families C₁–triazines, triazinones, uracilis, pyridazinone, phenyl–carbamates; C₂–ureas, amide; C₃–nitriles, benzothiadiazole and phenyl–pyridazine (Hrac 2004). Triazine resistance is the most prevalent type of herbicide resistance found in weed (Le Baron 1991).

Triazines

Ryan (1970) reported that a population of *Senecio vulgaris* was not controlled by the recommended rates of simazine in a conifer nursery. Since this report, resistance to PS II inhibitor herbicides has become widespread. Resistance of *Senecio vulgaris* to simazine, atrazine and other triazines was documented also by Radosevich and Appleby (1973). In 1992 the resistant biotype was found in an apple orchard in the Czech Republic (Chodová et al., 1993, 1995). Resistance in *Amaranthus hybridus* first occurred in Maryland in 1972 (Ritter and Menbere

1997), in *A. powellii* in Canada in 1977, in *A. retroflexus* in Canada in 1979 (Warwick and Weaver 1980) and in Hungary (Hartmann 1979).

Triazine resistant weed biotypes have frequently and independently occurred in many localities all over the world (Le Baron and Gressel 1982). Thus, triazine resistance in *Chenopodium album* occurred in 10 different countries (Le Baron and Gressel 1982), and in 17 states within the USA (Bandeem and McLaren 1976). *Amaranthus retroflexus* and *Chenopodium album* were widespread in areas with intensive maize and sugar beet growing and in orchards (Mikulka and Chodová 2000).

In 1976, resistant populations of *Polygonum lapathifolium* spread in France (Darmency et al., 1981). Populations of *P. lapathifolium* resistant to atrazine were found at a railway station in the Czech Republic, where atrazine had been applied at high rates for over 15 years (Mikulka and Chodová 2000). A biotype of *Solanum nigrum* resistant to atrazine was detected in France in 1979 (Gasquez et al., 1981).

Johnston and Wood (1976) confirmed triazine resistant *Kochia scoparia* from several locations at a railroad. *Kochia scoparia* resistant to atrazine was proved by us in 1998 at and close to railroad stations. This weed had been linearly dispersed for tens of thousands of kilometers along railroads (Gressel 2002). Triazine resistant *Poa annua* has appeared in France on one simazine treated roadside where the herbicide had been used once a year for a long time (Ducruet and Gasquez 1978). Resistance of *Coryza canadensis* to atrazine or simazine has been reported from tree orchards, vineyards, non-agricultural lands and railways. Mukulas and Pölös (1983) showed cross resistance to some phenylureas, carbamates and uracilis in Hungary; cross resistance of simazine resistance *C. canadensis* to diquat and paraquat was also confirmed in the Czech Republic (Mikulka and Chodová 1998).

Eleven weeds resistant to triazine herbicides were detected in Israel, e.g. *Alopecurus myosuroides*, *Lolium rigidum*, *Poa annua*, *Amaranthus hybridus*, *Chenopodium album* and *Coryza canadensis* (Rubin 1997).

Some resistant biotypes of weed species expanded over large areas (e.g. *Chenopodium album*, *Solanum nigrum*, *Senecio vulgaris*, *Poa annua*, *Amaranthus retroflexus*, *Coryza canadensis* and *Echinochloa crus-galli*) in Europe (De Prado et al., 1997). This contrasts with others that were found only in a specific area, e.g. *Bidens tripartita* in Austria (Szith and Furlan 1979) and *Amaranthus albus* and *A. blitoides* in Spain (De Prado et al., 1997).

Gressel (2002) has used a ranking system and the resistant weed database by HEAP to identify the first 15 of the most widespread triazine resistant weed species. They are: *Chenopodium album*, *Senecio vulgaris*, *Amaranthus hybridus*, *A. retroflexus*, *Kochia scoparia*, *Solanum nigrum*, *Ambrosia artemisiifolia*, *Poa annua*, *Echinochloa crus-galli*, *Lolium rigidum*, *Amaranthus powellii*, *Chenopodium strictum*, *Coryza canadensis*, *Polygonum lapathifolium* and *P. persicaria*.

The occurrence of further triazine resistance in weed species was presumably much delayed because farmers have used other alternative herbicides (Van Oorschot 1991).

Phenylureas

An example of how resistance to phenylurea arose is the selection of resistance biotypes through continuous treatment by chlorotoluron, resulting in the first grass biotype resistant to phenylurea in Europe (Germany) in *Alopecurus myosuroides* (Niemann and Pestemer 1984). Similar biotypes occurred e.g. in England (Moss 1987), France (Chauvel and Gasquez 1990; Letouze et al., 1997), Spain (De Prado et al., 1991), the Netherlands (Van Oorschot and Van Leeuwen 1992) and in Bulgaria (Chipeva and Nikolova 2004). *Apera spica venti* resistant to isoproturon was found in Switzerland (Mayor and Maillard 1997) and in Germany (Niemann 2000).

The number of weed species resistant to phenylureas did not rise, but new locations have been documented in many countries. Some populations of *Alopecurus myosuroides*, *Avena fatua* and *Lolium rigidum* showing cross-resistance to multiple classes of herbicides, e.g. acetolactate synthase and acetyl-CoA carboxylase inhibitors, were described.

Weed resistance to acetolactate synthase inhibitors

Weed species acquired resistance to acetolactate synthase (ALS) inhibitors faster than to any other group of herbicides. This is due to the fact that all the sulfonylurea and imidazolinone herbicides had a high persistence (Gressel 2002).

Chlorsulfuron was registered to control weeds in North America in 1982. Chlorsulfuron resistant *Lactuca serriola* was discovered in 1987 (Mallory-Smith et al., 1990) and *Kochia scoparia* in 1989 (Primiani et al., 1990). *Kochia scoparia* resistant to ALS inhibitors is now present in 18 states of the USA.

Very important and widespread resistant weeds are also *Ambrosia artemisiifolia*, *A. trifida* and *Amaranthus retroflexus* (Ballard et al., 1995; McNaughton et al., 2001). Documented occurrences of resistance to ALS inhibitors in Canada include *Stellaria media* (O'Donovan et al., 1994a), *Kochia scoparia* (Morrison and Devine 1994) and *Sonchus asper* (Rashid et al., 2003). *Raphanus raphanistrum* resistant to ALS inhibitors was detected by Yu et al. (2003) in Australia. ALS inhibitors have been widely used also in Europe and the repeated uses of sulfonylureas in crops and imazapyr on railways were soon followed by resistance. A biotype resistant to sulfonylureas has been detected in *Stellaria media* in Denmark (Kudsk et al., 1995), in *Papaver rhoeas* in Spain (Claude et al., 1998), Italy and Greece (Claude and Cornes 1999), and in a population of *Alopecurus myosuroides* and *Lolium rigidum* in England (Moss and Cussans 1991). Several weed biotypes resistant in Israel: *Amaranthus blitoides* and *Amaranthus retroflexus* (Rubin et al., 1992; Sibony and Rubin 1999), and *Conyza canadensis* (Lior et al., 1994).

Weed resistance to acetyl-CoA carboxylase inhibitors

Resistance to acetyl-CoA carboxylase (ACCCase) inhibitors developed in grass weeds. Resistant biotypes have been detected in Europe (Germany, England, Spain, France, Belgium, the Netherlands, Greece and Turkey) in winter wheat fields where graminicides had been used continuously. The resistant biotypes

belong to *Avena fatua* (Joseph et al., 1990), *A. sterilis* (Mansooji et al., 1992), *Lolium rigidum* (Cotterman and Saari 1992), *L. multiflorum* (Gronwald et al., 1992) and *Alopecurus myosuroides* (Lainsbury 1998). Populations of *Sorghum halepense* resistant to fluazifop and quizalofop were also detected in the USA (Barrentine et al., 1992).

Weed resistance to inhibitors of microtubules

α - and β -Tubulin form the core building blocks of the tubulin polymers that comprise spindle fibres as the key elements in chromosome alignment and separation during cell division. Several groups of herbicides, including the dinitroanilines and carbamates, block tubulin polymerisation by binding to tubulin monomers (Devine and Preston 2000). Populations of *Eleusine indica* resistant to trifluralin (chemical family dinitroanilines) were discovered in the USA in fields where trifluralin had been used for 10 years (Mudge et al., 1984) and resistant *Stellaria viridis* in cereals and oilseed crops in Canada (Morrison et al., 1989). Dinitroaniline resistance is conferred by a mutation in the α -tubulin gene (Anthony et al., 1998).

Weed resistance to auxin analog herbicides

Active ingredients from the chemical family phenoxy-carboxylic acids such as 2,4-D and MCPA are among the oldest synthetic herbicides. These compounds are used for postemergence management of dicot weeds in grass crops, pastures and lawns. The tolerance of grasses to these herbicides appears to be related to rapid irreversible metabolic conversion to non-toxic products, whereas in dicotyledonous species the herbicide is often found in the form of reversible conjugates. At low rates, these substances act similarly to the natural auxin indole acetic acid. The precise mechanism of action of these herbicides is still not completely understood. Research along various lines, including herbicide resistant mutants, supposed that these herbicides disrupt the perception of the natural auxin signal, or reduce it artificially (Sterling and Hall 1997). The auxin receptor had lost its affinity to bind phenoxy herbicides in the *Sinapsis arvensis* mutant (Deshpande and Hall 2000; Hall and Zheng 2000).

Heap and Morrison (1992) found populations of *Sinapsis arvensis* in Canada which were resistant to 2,4-D and MCPA. Biotypes of *Cirsium arvense* resistant to 2,4-D were detected in Sweden (Fogelfors 1979). *Stellaria media* resistant to mecoprop was found in England (Coupland et al., 1990). After over 20 years of treatment by auxin-like herbicides, a *Papaver rhoeas* biotype resistant to 2,4-D has been found in Spain (Taberner et al., 1996). Only the grass weed *Echinochloa crus-galli* has been found to be tolerant to 2,4-D and quinclorac, the active ingredient of quinoline carboxylic acids in Europe (Coupland 1994; López-Martínez et al., 1995).

Weed resistance to inhibitors of biosynthesis of carotenoides

This chemical family of herbicides (triazole) are known as bleaching herbicides. A biotype of *Lolium rigidum* resistant to triazoles was found in Australia

in 1988. Research has shown that these biotypes are resistant to the active ingredient amitrole and may be cross-resistant to other herbicides (Burnet et al., 1992). Resistant populations of *Poa annua* and *Polygonum aviculare* were detected in apple orchards in Belgium after application of amitrole (Bulcke et al., 1988).

**Weed resistance to inhibitors of
5-enol-Pyruvyl-shikimate-3-phosphate synthase**

Glyphosate inhibits the enzyme 5-enol-Pyruvyl-shikimate-3-phosphate synthase (EPSP), a key enzyme in the shikimate pathway leading to the biosynthesis of phenylalanine (e.g. Devine and Preston 2000). Glyphosate has only low selection pressure on weed populations. Since the early 1990's, glyphosate resistance has been transferred into several crop species (e.g. maize, soybean, tobacco, sugar beet, rape) by genetic engineering. The first confirmed cases of glyphosate resistance were in *Lolium rigidum* biotypes in Australia after application of glyphosate over 15 years (Powles et al., 1998). Within 3 years of using only glyphosate for weed control in continuous cropping with glyphosate resistant soybeans, glyphosate failed to control *Conyza canadensis* in some fields (Van Gressel 2001). Glyphosate resistance in an *Eleusine indica* population appears to be due to a mutation in EPSP (Tran et al., 1999).

**Herbicide Resistance in Europe
Compared With the Rest of the World**

This section is an updated summary of a review presented at 13th Australian Weeds Conference (Moss 2002). Herbicide-resistant weeds have been identified in 21 European countries with the highest number of resistant biotypes found in France (30), Spain (26), United Kingdom (24), Belgium (18) and Germany (18). Consequently, herbicide-resistance appears to be mainly a problem in Western-Europe, although occurrence in Eastern Europe appears to be increasing but is less well documented. These 21 European countries represent 36% of the 59 countries world-wide in which herbicide resistant weeds have so far been detected (Heap 2003).

Resistance has evolved in 55 species in Europe and those in which resistance has evolved most frequently are listed in Tab. 2.

While there is no clear relationship between plant families or genera and their tendency to evolve resistance, grass-weeds tend to be over-represented in the list of resistant biotypes both within Europe and world-wide. While grass-weeds account for 33% of all resistant species and 40% of all resistant biotypes, they account for only 25% of the world's major weeds (Heap 1999). Within Europe, the major resistance problems currently are the grass-weeds black-grass (*Alopecurus myosuroides*) and rye-grass (*Lolium* spp.).

Tab. 2. The 15 commonest herbicide-resistant weed species in Europe in terms of the number of countries in which resistant biotypes have been recorded (based on Heap 2003)

	Weed species	Countries
1	Fat-hen (<i>Chenopodium album</i>)	14
2	Pigweeds (<i>Amaranthus</i> spp., mainly <i>retroflexus</i>)	10
3	Black Nightshade (<i>Solanum nigrum</i>)	10
4	Groundsel (<i>Senecio vulgaris</i>)	8
5	Black-grass (<i>Alopecurus myosuroides</i>)	7
6	Canadian Fleabane (<i>Conyza canadensis</i>)	7
7	Annual Meadow-grass (<i>Poa annua</i>)	7
8	Cockspur (<i>Echinochloa crus-galli</i>)	7
9	Polygonums (<i>Polygonum</i> spp.)	6
10	Common Chickweed (<i>Stellaria media</i>)	6
11	Rye-grasses (<i>Lolium</i> spp.)	5
12	Willowherbs (<i>Epilobium</i> spp.)	5
13	Wild-oats (<i>Avena</i> spp.)	4
14	Common Poppy (<i>Papaver rhoeas</i>)	4
15	Bristle-grasses (<i>Setaria</i> spp.)	3

Within Europe, triazine-resistance occurs in 16 countries, ALS resistance in 14 countries, urea/amide resistance in 11 countries, ACCase resistance in eight countries and resistance to other mode of action groups in seven countries. Tab. 3. relates the proportion of resistant biotypes to each of the major mode of action groups for the 21 European and 38 non-European countries with resistance.

Tab. 3. Proportion (%) of total resistant biotypes in 21 European and 38 non-European countries related to herbicide mode of action (based on Heap 2003)

	Herbicide mode of action group (Schmidt 1997)					Total (%)
	A ACCcase inhibitors	B ALS inhibitors	C1 Triazines	C2 Ureas/ Amides	Other	
European countries	9	12	61	9	9	100
Non-European countries	16	33	16	7	28	100
World-wide	13	25	34	8	20	100

These % values allow comparison of the relative incidence of resistance to different mode of action groups in Europe compared with countries in the rest of the world. Compared with the 38 non-European countries in which resistance has developed, resistance to ALS inhibitors tends to be less prevalent (12% vs. 33%), and resistance to triazines more prevalent (61% vs. 16%) in Europe. The incidence of resistance to ACCase inhibitors and ureas/amides is broadly similar in European and non-European countries.

In summation, 91% of cases of resistance in Europe are associated with just four herbicide mode of action groups: ACCase and ALS inhibitors, and

triazine and urea/amide photosynthetic inhibitors. In the 38 non-European countries, the comparative figure is 72%, showing that resistance to other herbicide groups is comparatively more important. There are also a few cases in Europe of resistance to bipyridiliums, dinitroanilines and synthetic auxins. While resistance to ALS inhibitors is currently less prevalent in Europe than elsewhere, it is likely to increase. Indeed, there are indications that this is happening. Six of the nine new cases of resistance recorded since publication of a previous review of herbicide resistance in Europe (Moss 2002), involve ALS inhibitors.

Mechanism and Molecular Basis of Resistance

Herbicides kill plants by disrupting essential physiological or biochemical processes, usually through a specific interaction with a single molecular target in the plant. The herbicide molecules, after delivery to the target cells or tissues, inhibit these processes so that the plant can no longer survive (Devine and Preston 2000). Herbicide resistance can be conferred by several mechanisms, the most important of which are target site insensitivity, an altered binding site and rapid metabolic transformation of the herbicide to inactive products. Other potential resistance mechanisms include reduced uptake into the cells (Lutman and Heath 1990).

Resistance based on target site modification

Reduced sensitivity of the target site is the most common resistance mechanism in herbicide resistant weeds. Target sites are usually enzymes, proteins or other components in the plant where herbicides bind and thereby disrupt normal plant function. The number of target sites is between 15 and 20 (Cobb and Kirkwood 2000). The consequence of such a limited number of target sites is that resistance to existing herbicides is becoming increasingly prevalent. Target site-based resistance is usually conferred by a mutation in the target protein that decreases herbicide binding without compromising the function of the protein (Devine and Eberlein 1997). This has been documented for herbicides that target most major known sites of action, including D1 protein, acetolactate synthase and acetyl-CoA carboxylase (Devine and Shukla 2000).

Herbicides binding to target sites are presented in Tab. 4.

The following part of the review presents the physiological and molecular basis of target site-based resistance to herbicides affecting the major known sites of action.

Tab. 4. The sites of herbicides action

Target site	Process inhibited	Representative chemical groups	Total number of resistant weed biotypes
D1 (Q _B) protein	photosynthetic electron transport	s-triazines, phenylureas, uracilis	86
PS I electron acceptor	photosynthetic electron transport	bipyridiliums	22
Phytoene desaturase	carotenoid biosynthesis	various	2
Protoporphyrinogen oxidase	porphyrin biosynthesis	nitro-diphenylethers, oxadiazon	2
Acetolactate synthase	branched-chain amino acid biosynthesis	sulfonylureas, imidazolinones, triazolopyrimidines	86
enol-Pyruvyl-shikimate-3-phosphate synthase	aromatic amino acid biosynthesis	glyphosate	6
Acetyl-CoA carboxylase	fatty acid biosynthesis	cyclohexanediones, aryloxyphenoxypropionates	34
„Elongase“ complex	fatty acid elongation	thiocarbamates	8
α-, β-Tubulin	cell division	dinitroanilines, carbamates, phosphoric amides	10
Auxin-binding protein	multiple	phenoxyacetic acids, benzoic acids	24

From Devine and Preston (2000) and Heap (2004)

D1 (Q_B) protein

Triazines (e.g. atrazine, simazine), phenylureas (e.g. chlorotoluron, isoproturon, diuron) and uracil herbicides (lenacil) inhibit photosynthesis electron transport in PS II by binding to the D1 protein and blocking the transport by the mobile electron carrier plastoquinone Q_B (e.g. Trebst et al., 1988, Trebst 1991; Holá et al., 2004).

The most common mechanism of resistance to triazine herbicides is target site mutation in the *psbA* gene which codes for the D1 protein (Golden and Haselkorn 1985). Molecular analysis shows that in most cases resistance is due to a Ser₂₆₄ to Gly mutation (Shukla and Devine 2000).

Ernst et al. (1996) found that some sensitive *Senecio vulgaris* biotypes had a Gly residue at position 264, but that two other mutations, Ala₂₅₁ to Arg and Val₂₈₀ to Leu, were present in resistant biotypes. Resistance to triazine herbicides can be conferred by amino acid substitution at position Val₂₁₉ in *Poa annua* biotypes that are resistant to diuron and metribuzin (Mengistu et al., 2000).

Acetolactate synthase

Chlorsulfuron as well as other sulfonylureas and imidazolinone herbicides (e.g. imazapyr) are effective at low rates, which is related to their highly specific inhibition of the acetolactate synthase (ALS) (Ray 1984; Saari et al., 1994). Inhibition of ALS leads to the starvation of the weeds for the branched-chain amino acids isoleucine, valine and leucine, and it is the primary mechanism by which ALS-inhibiting herbicides cause the death of weed plants (Rathinasabapathi et al., 1990). ALS-inhibitors have been widely used since their introduction in the early 1980's and now constitute one of the major mode-of-action groups used (Devine and Shukla 2000).

Target site based resistance to ALS-inhibition is due to point mutation that occurs within discrete conserved domains of the ALS gene (Devine and Eberlein 1997). A mutation to resistance at the position Pro₁₉₇ was identified in *Lactuca serriola*, *Kochia scoparia* and *Amaranthus retroflexus*; mutation at position Ala₁₂₂ in *Xanthium strumarium*, *Amaranthus hybridus* and *Solanum ptycanthum*; mutation at position Ala₂₀₅ in *Xanthium strumarium*, *Amaranthus hybridus*, *A. rudis*, *Kochia scoparia* and *Ambrosia artemisiifolia*; and mutation at position Ser₆₅₃ in *Amaranthus powellii*, *A. retroflexus* and *A. rudis* (Tranel and Wright 2002).

The frequency of spontaneous chlorsulfuron resistance and imidazolinone resistance through ALS mutations in *Arabidopsis thaliana* is estimated to be approximately 1×10^{-9} (Saari et al., 1994).

Acetyl-CoA carboxylase

Cyclohexanediones (CHD) and aryloxyphenoxy-propionates (AOPP) have become important for the control of grass weeds in a variety of broadleaf and cereal crops. These herbicides catalyse the first step in fatty acid biosynthesis. Inhibition of acetyl-CoA carboxylase (ACCase) leads to inhibition of acyl lipid biosynthesis (Burton et al., 1987).

Dicot plastids contain the prokaryotic form of ACCase (multi-subunit, coded by three separate genes) which is insensitive to AOPP and CHD herbicides, and the eukaryote form (herbicide sensitive) is extra-plastidic. The plastids of monocots contain the eukaryotic form of ACCase (one protein with three functional domains) which is sensitive (Devine and Eberlein 1997; Devine and Shulka 2000). Monocots lack the prokaryotic form due to the loss of the accD gene in the chloroplast genome. Multiple forms of eukaryotic ACCase are present in some grasses that differ in herbicide sensitivity (Konishi and Sasaki 1994; Devine and Shulka 2000). This is the primary basis for selectivity of these herbicides between grasses and dicots (Devine and Shulka 2000).

Resistance of some grass species or cereal crops is based on enhanced metabolic degradation of herbicides to inactive compounds (Devine and Shimabukuro 1994; Cocker et al., 1999). Resistance due to an alteration in the target enzyme, making it less sensitive to inhibition by these herbicides, has been documented in many species including: *Avena fatua*, *A. sterilis*, *Lolium rigidum*, *Eleusine indica*, *Setaria viridis*, *S. faberi* and *Alopecurus myosuroides*. It appears that

several different mutations can occur, each conferring a unique pattern of cross-resistance to cyclohexanediones and aryloxyphenoxypropionates (e.g. Marles et al., 1993; Tardif and Powles 1993; Letouze and Gasquez 2000).

Resistance due to increased herbicide metabolism

For several weed biotypes it has been reported that their resistance to a herbicide is due either to rapid degradation or conjugation of toxic compounds (Kemp et al., 1990; Preston and Powles 1997; De Prado et al., 1998; Devine and Preston 2000) (see Tab. 5.).

Among the enzymatic systems thought to be responsible for such resistance are glutathione-S-transferases and cytochrome P450 monooxygenase. Rapid herbicide detoxification is also important for herbicide tolerance of crops and allows selective use of herbicides to control weeds without damage to the crops (Cole 1994). We hereinafter list several examples of enzymes responsible for herbicide resistance due to an increased metabolism of herbicides.

Tab. 5. Weed species with herbicide resistance due to increased metabolism of herbicide

Weed species	Herbicides
<i>Alopecurus myosuroides</i>	Chlorotoluron Pendimethalin Diclofop-methyl Fenoxaprop-P-ethyl Propaquizafop Chlorsulfuron
<i>Apera spica venti</i>	Chlorotoluron
<i>Abutilon theophrasti</i>	Atrazine
<i>Avena sterilis</i>	Diclofop-methyl
<i>Bromus tectorum</i>	Chlorotoluron
<i>Digitaria sanguinalis</i>	Fluazifop-P-butyl
<i>Echinochloa colona</i>	Propanil
<i>Echinochloa crus-galli</i>	Propanil
<i>Hordeum leporinum</i>	Fluazifop-P-butyl
<i>Lolium rigidum</i>	Simazine Diclofop-methyl Fluazifop-P-butyl Chlorsulfuron Metribuzin Chlorotoluron
<i>Sorghum halepense</i> L. (Pers.)	Bentazon
<i>Stellaria media</i>	Mecoprop

From Devine and Preston (2000)

Glutathione-S-transferases

Glutathione-S-transferases belong to the group of enzymes responsible for the metabolism of a number of herbicides. Enhanced atrazine detoxification in the resistant populations of *Abutilon theophrasti* is mediated by glutathione-S-transferases (Gray et al., 1995). These enzymes have also been found to play a role in the resistance of e.g. *Alopecurus myosuroides* to fenoxaprop-P-ethyl (Cummins et al., 1997) and to a range of herbicides from different chemical groups (Milner et al., 1999). An increased export of herbicide conjugates with glutathion or glucose may play a role in the resistance of *Digitaria sanguinalis* to fluazifop-P-buthyl (Hidayat and Preston 1997).

Cytochrome P450 monooxygenases

Cytochrome P450 monooxygenases represent the large family of enzymes responsible for the oxygenation of hydrophobic substrates in plants (Bolwell et al., 1994). Cytochrome P450 monooxygenases catalyse diverse reaction types, but the most common reactions with herbicides as substrates are hydroxylations and dimethylations (Cole 1994; Devine and Preston 2000). Populations of several grass species (e.g. *Stellaria media*, *Lolium rigidum*, *Avena sterilis*, *Alopecurus myosuroides* and *Phalaris minor*) have evolved resistance to herbicides as a result of enhanced cyt P450-dependent detoxification (Burnet et al., 1993; Carrera et al., 1999; Cocker et al., 1999).

Gene Flow

Gene flow, via pollen or seed, from a field with herbicide resistant plants to an adjacent or nearby field with susceptible plants may provide an initial source of genes for resistance for the evolution of herbicide resistance (Jaseniuk et al., 1996). Pollen mediated gene flow of herbicide resistance genes depends on distance, pollen characteristics, abundance of pollen, mating system and environmental conditions (Darmency 1996).

With one exception, resistance to all classes of herbicides examined to date is determined by nuclear inheritance. Resistance to triazines is inherited cytoplasmically in most of the weed species (Jaseniuk et al., 1996). The gene conferring triazine resistance is located in the chloroplast genome (Holliday and Putwain 1980). The atrazine tolerance in *Abutilon theophrasti* was not cytoplasmically inherited (Andersen and Gronwald 1987).

If resistance is due to an enhanced metabolism of herbicides, plants having weak resistance mechanisms have some chance to survive and therefore contribute to the next generation. If resistance is due to target site resistance, the enrichment of resistance genes in surviving weeds may lead to individuals expressing multiple resistance. Large population size, herbicide selection pressure and a rapid turnover of generations are required for such a process (Darmency 1996). Pollen grains have been collected in *Kochia scoparia* in field experiments up to 62 m from the closest pollen source (Mulugeta et al., 1992).

Although pollen dispersion has generally been assumed to be the major mechanism of interpopulation gene flow in plants, seed dispersion may be most important in weed populations (Jaseniuk et al., 1996). Most weeds resistant to herbicides belong to so-called colonising and pioneering species. They produce a large number of seeds per plant (e.g. *Amaranthus retroflexus*: 500 000, *Conyza canadensis*: 60 000; *Chenopodium album*: 3 000). Furthermore, some weeds can have several generations per year, like *Senecio vulgaris* or *Stellaria media*.

Seeds are spread through cultivation and harvest machines. The use of contaminated manure resulted in new infestations with resistant biotypes in areas never treated with herbicides. Some animals are responsible for spread of seeds nearby, while wind can move seeds over large distances (Jaseniuk et al., 1996).

Conclusions

For deciding on the direction of weed management strategy in future it is important to know how herbicide resistance in weed populations evolved. Grass weed (*Alopecurus myosuroides*, *Lolium multiflorum*, *L. rigidum*, *Avena fatua*, *Apera spica-venti*, *Phalaris paradoxa* and *Bromus sterilis*) and broadleaf weeds (*Papaver rhoeas*, *Matricaria* spp., *Stellaria media*) have developed resistance to various herbicides used in wheat production in Europe. The problem of weed resistance to the currently used herbicides is a major issue as resistant weeds exhibit resistance to multiple herbicides and alternative herbicides that provide effective control are not available (Matthews and Powles 1992; Preston et al., 1996; Gianessi et al., 2003).

The cooperation of scientists, producers of herbicides and farmers is necessary both for the successful detection of resistant biotypes of weeds (Moss et al., 1999) and for effective prevention of resistance. These aspects are especially important in the development of new herbicide substances and if growing technologies involving genetically modified crops are introduced.

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REZISTENTNOST KOROVA NA HERBICIDE – MEHANIZMI I MOLEKULARNE OSNOVE

- pregledni rad -

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Izvod

Rezistentnost korova na herbicide raste pratećim koracima u svetu. Ovaj problem je obično rezultat velikog selekcionog pritiska na korovsku populaciju usled primene visokih doza rezidualnih herbicida, gajenja useva u monokulturi i minimalne obrade zemljišta. Kao odgovor na visok selekциони pritisak, usled uzastopne primene herbicida sa istim mehanizmom delovanja menja se genetički sastav korovskih populacija, tako što se povećava frekvencija rezistentnih alela i individua. Ponovnom primenom herbicida istog ili sličnog mehanizma delovanja uništavaju se osetljive populacije, dok rezistentne preživljavaju, razmnožavaju se i prenose osobine rezistentnosti na sledeću genetaciju.

Masovna pojava rezistentnih korovskih biljaka može da bude glavni ograničavajući faktor dalje primene herbicida, jer je za uništavanje rezistentnih biotipova pojedinih korovskih biljaka zavisno od stepena rezistentnosti neophodno primeniti veće količine herbicida. Povećana primena herbicida po jedinici površine dovodi do poskupljenja proizvodnje, povećane kontaminacije životne sredine i potreba za iznalaženjem novih herbicida sa specifičnim ili drugačijim mehanizmom delovanja.

U radu je dat veoma detaljan prikaz sadašnjeg stanja distribucije rezistentnosti korovskih biljaka na herbicide, kao i mehanizmi i molekularne osnove rezistentnosti.