Origin, Mechanism and Molecular Basis of Weed Resistance to Herbicides

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Abstract

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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. Czech biotypes of resistant weeds with a mutation at codon 264 of the *psbA* gene encoding the D1 protein and at codon 574 of the acetolactate synthase gene are presented.

Keywords: resistance to herbicides; origin and spread of resistant weeds; target site; metabolic resistance; molecular basis of resistance

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 1988; Chodová *et al.* 1989; Mikulka & Chodová 1990, 1992a,b, 1995, 1998; Chodová & Mikulka 1991a,b, 2000c; Chodová & Salava 2004b; Salava *et al.* 2004b). 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

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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 & 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, sulfonylureas and imidazolinones have often been used continuously without any rotation of crops and herbicides (De Prado *et al*. 1997). The poor agricultural practice of monoculture has led to soil and environmental problems, uncontrolled insects and diseases, and herbicide resistance (e.g. Gressel 1997; Mikulka & Chodová 2000). Until now, 291 resistant biotypes belonging to 174 species (104 broadleaf and 70 grass weeds) have been identified as resistant to various herbicides (Heap 2004). The numbers of herbicide resistant weed species differ with the area (Table 1). 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).

Resistance often develops in species that have a relatively rapid turnover of their seed bank. This turnover is either characteristic of the genus, as with *Kochia*, *Chenopodium* and *Amaranthus*, or was aided by the cultivation practices. One of the latter is the absence of cultivation to control weeds, particular moldboard plowing (Ball 1992). A practice that seems to be related to the development of triazine resistance is the application of manure to fields which will add seed back to the field (Ritter 1989; Cifera *et al.* 1992).

ORIGIN 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

Table 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	ACCase inhibitors	ALS inhibitors	Triazines	Ureas/ amides	Bipyridiliums	Dinitroanilines	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

From Heap (2004)

C_1-triazines, triazinones, uracils, pyridazinone, phenyl-carbamates; C_2-ureas, amide; C_3-nitriles, benzothiadiazole and phenyl-pyridazine (Hrac 2004). Triazine resistance is the most prevalent type of herbicide resistance found in weeds (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 & Mikulka 1992, 1995; Chodová *et al.* 1993, 1995).

Resistance in *Amaranthus hybridus* first occurred in Maryland in 1972 (Ritter & Menbere 1997), in *A. powellii* in Canada in 1977, in *A. retroflexus* in Canada in 1979 (Warwick & Weawer 1980) and in Hungary (Hartmann 1979). Triazine resistant weed biotypes have frequently and independently occured in many localities all over the world (Le Baron & Gressel 1982). Thus, triazine resistance in *Chenopodium album* occurred in 10 different countries (Le Baron & Gressel 1982), and in 17 states within the USA (Bandeen & McLaren 1976).

Amaranthus retroflexus and *Chenopodium album* were widespread in areas with intensive maize and sugar beet growing and in orchards (Zemánek & Mikulka 1985; Mikulka 1988; Mikulka & Chodová 1987, 1992b, 1996, 1998).

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 *et al.* 1988), and resistant *P. persicaria* was found at a railway station in 1989 (Mikulka & Chodová 1996, 1998).

Populations of *Chenopodium strictum* resistant to atrazine have appeared in corn and sugar beet fields in the Czech Republic in 1989 (Mikulka & Chodová 1990). One atrazine resistant population of *C. pedunculare* was found in corn (Mikulka & Chodová 1998).

A biotype of *Solanum nigrum* resistant to atrazine was detected in France in 1979 (Gasquez *et al.* 1981); in the Czech Republic it was found at a railway station where this herbicide had been applied for 5 years (Mikulka & Chodová 1998; Salava *et al.* 2004b).

Failure of triazine herbicides to control *Ambrosia artemisiifolia* and *Brassica campestris* in a field was first reported in 1976 (Souza Machado *et al.* 1977, 1978).

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 (Chodová & Mikulka 2000a,b). This weed had been linearly dispersed for tens of thousands of kilometeres 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 & Gasquez 1978). *Poa annua* resistant to simazine was found in apple orchards in North Bohemia after long-time application of simazine (Chodová *et al.* 1994), and resistant *Setaria viridis* was found on railroads and in corn (Mikulka & Chodová 1996, 1998).

Resistance of *Conyza canadensis* to atrazine or simazine has been reported from tree orchards, vineyards, non-agricultural lands and railways. Mikulas and Pölös (1983) showed cross resistance to some phenylureas, carbamates and uracils in Hungary; cross resistance of simazine resistant *C. canadensis* to diquat and paraquat was also confirmed in the Czech Republic (Chodová *et al.* 1987; Mikulka & 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 *Conyza 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, Conyza 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 & Furlan 1979) and *Amaranthus albus* and *A. blitoides* in Spain (De Prado *et al.* 1988).

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 artemi-*

siifolia, *Poa annua*, *Echinochloa crus-galli*, *Lolium rigidum*, *Amaranthus powellii*, *Chenopodium strictum*, *Conyza canadensis*, *Polygonum lapathifolium* and *P. persicaria*.

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

Phenylureas

An example for how resistance to phenylurea arose is the selection of resistant biotypes through continuous treatment by chlorotoluron. This was responsible for the first grass biotype resistant to phenylurea in Europe (Germany) in *Alopecurus myosuroides* (Niemann & Pestemer 1984). Similar biotypes appeared e.g. in England (Moss 1987), in France (Chauvel & Gasquez 1990; Letouze *et al.* 1997), in Spain (De Prado *et al.* 1991; Mendezez *et al.* 1994), in the Netherlands (Van Oorschot & Van Leeuwen 1992) and in Bulgaria (Chipeva & Nikolova 2004). *Apera spica venti* resistant to isoproturon was found in Switzerland (Mayor & Maillard 1997) and in Germany (Niemann 2000).

Extensive and repeated use of phenylurea herbicides has resulted in the evolution of a resistant biotype of *Phalaris minor* in India (Singh *et al.* 1997).

Different *Echinochloa crus-galli* populations resistant to propanil have appeared in a rice field in Greece (Giannopolitis & Vassilou 1989). Carey *et al.* (1995) concluded that propanil resistance in *Echinochloa crus-galli* from Arkansas was not due to differential absorption, translocation or modification at the site of action of the herbicide.

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.

Weeds resistant 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, Salsola iberica* (Morrison & Devine 1994), *Galium spurium* (Hall *et al.* 1998) 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 & Cornes 1999), and in a population of *Alopecurus myosuroides* and *Lolium rigidum* in England (Moss & Cussans 1991; Christopher *et al.* 1991).

The occurrence of *Kochia scoparia* resistant to ALS inhibitors in the Czech Republic is closely related to imazapyr applications to control weeds on the railroad (Chodová & Mikulka 1997, 1998, 2000a,d, 2001; Mikulka & Chodová 2000, 2002). Kochia plants showed cross-resistance to other sulfonylureas such as nicosulfuron, triflusulfuron-methyl, tribenuron, prosulfuron + primisulfuron and chlorsulfuron (Chodová & Mikulka 2000b).

Several weed biotypes resistant to ALS inhibitors have also been detected in Israel: *Amaranthus blitoides* and *Amaranthus retroflexus* (Rubin *et al.* 1992; Sibony & Rubin 1999), *Conyza canadensis* (Lior *et al.* 1994) and *Cuscuta campestris* (Sibony *et al.* 1995).

Weeds resistant to acetyl-CoA carboxylase inhibitors

Resistance to acetyl-CoA carboxylase (ACCase) 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 & Saari 1992), *L. multiflorum* (Gronwald *et al.* 1992) and *Alopecurus myosuroides* (Lainsbury 1998).

A biotype of *Lolium multiflorum* resistant to diclofop was detected in the USA (Stanger & Appleby 1989), of *Avena fatua* in Canada and the USA (Heap *et al.* 1993; Seefeldt *et al.* 1994) and of *Setaria viridis* in Canada (Marles *et al*. 1993). Populations of *Sorghum halepense* resistant to fluazifop and quizalofop were also detected in the USA (Barrentine *et al.* 1992).

Weeds resistant to a group of Photosystem I disrupting herbicides

The bipyridilium herbicides paraquat and diquat inhibit the photosynthetic electron transport in Photosystem I (PS I) by diverting electrons from one of the iron-sulphur carriers, possibly ferredoxin (Preston 1994). Resistance to paraquat was detected in several countries, mainly in perennial crops, orchards and plantations where repeated application of herbicide was practiced. Regardless of the fact that these herbicides have been used extensively for over 40 years, resistance has only evolved in populations of 25 species (Heap 2004). The use of bipyridil herbicides in orchards has selected resistant biotypes of *Conyza canadensis* e.g. in Hungary (Pölös *et al.* 1988) and in the Czech Republic (Mikulka & Chodová 1998), and in *Epilobium ciliatum* and *Poa annua* in England (Putwain 1982; Clay 1989).

There is no evidence that either a target site alteration to PS I or metabolism of paraquat contribute to resistance (Devine & Preston 2000).

Weeds resistant 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 & 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 *Setaria viridis* in cereals and oilseed crops in Canada (Morrison *et al.* 1989). Resistant populations of *Alopecurus myosuroides* were detected in England (Moss 1990). Dinitroaniline resistance is conferred by a mutation in the α-tubulin gene (Anthony *et al.* 1998).

Weeds resistant 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 & Hall 1997). The auxin receptor had lost its affinity to bind phenoxy herbicides in the *Sinapis arvensis* mutant (Deshpande & Hall 2000; Hall & Zheng 2000).

Wild carrot populations resistant to 2,4-D were first reported by Switzer (1952). Heap and Morrison (1992) found populations of *Sinapis 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 (Barnwell & Cobb 1989; 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-Martínéz *et al.* 1995).

Weeds resistant to inhibitors biosynthesis of carotenoids

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 *Agrostis stolonifera*, *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*-Pyruvylshikimate-3-phosphate synthase

Glyphosate inhibits the enzyme 5-*enol*-Pyruvylshikimate-3-phosphate synthase (EPSP), a key enzyme in the shikimate pathway leading to the biosynthesis of phenylalanine (e.g. Devine & 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 Gessel 2001). Glyphosate resistance in an *Eleusine indica* population appears to be due to a mutation in EPSP (Tran *et al.* 1999).

Weeds resistant to inhibitors of biosynthesis of lipids

Several different classes of herbicides (e.g. thiocarbamates) have been reported to interfere with some aspects of lipid biosynthesis. Triallate was a highly successful thiocarbamate herbicide that specifically controls *Avena* species in cereal crops.

Triallate resistant biotypes of *Avena fatua* were reported in Canada and in the USA by O'Donovan *et al.* (1994b) and Kern *et al.* (1996).

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 & 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 & 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 & 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 & 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 & Shukla 2000).

Herbicides binding to target sites are presented in Table 2.

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.

D1 (Q_B) protein

Triazines (e.g. atrazine, simazine), phenylureas (e.g. chlorotoluron, isoproturon, diuron) and uracil herbicides (lenacil) inhibit photosynthetic electron transport in PS II by binding to the D1 protein and blocking the transport by the mobile electron carrier plastoqinone Q_B (e.g. Trebst *et al.* 1988; Kočová *et al.* 1988; Trebst 1991; Körnerová *et al.* 1998; Holá *et al*. 2004).

The most common mechanism of resistance to triazine herbicides is target site mutation in the *psb*A gene which codes for the D1 protein (Golden & Haselkorn 1985). Molecular analysis shows that in most cases resistance is due to a Ser_{264} to Gly mutation (Hirschberg & McIntosh 1983; Shukla & Devine 2000). This substitution was confirmed

Table 2. The sites of herbicide action

Target site	Process inhibited	Representative chemical groups	Total number of resistant weed biotypes
D1 (Q_B) protein	photosynthetic electron transport	s-triazines, phenylureas, uracils	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-Pyruvylshikimate-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)

in Czech biotypes of *Kochia scoparia* (Chodová & Salava 2004a), *Solanum nigrum* (Salava *et al.* 2004b) and further weeds as shown in Table 3.

Ernst *et al.* (1996) found that some sensitive *Senecio vulgaris* biotypes had a Gly residue at position 264, but that two other mutations, Ala_{251} to Arg and Val_{280} to Leu, were present in resistant biotypes.

Resistance to triazine herbicides can be conferred by amino acid substitution at position Val_{219} in *Poa annua* biotypes that are resistant to diuron and metribuzin (Mengistu *et al.* 2000).

The initial frequency of triazine resistant mutations has been estimated to be from 10^{-10} to 10^{-20} (Rubin 1991).

Acetolactate synthase

Chlorsulfuron as well as other sulfonylureas and imidazolinone herbicides (e.g. imazapyr) are effective at low rates; this 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 1980s, and now constitute

Table 3. Czech weed biotypes with the mutation at codon 264 of the *psbA* gene

Species	Active ingredient	Cross resistance
Amaranthus retroflexus	atrazine	atrazine, simazine, prometryn, terbutryn, cyanazine
Chenopodium album		atrazine, simazine, prometryn, terbutryn, terbuthylazine, cyanazine, chloridazon, lenacil
Solanum nigrum		simazine
Conyza canadensis	simazine	atrazine, prometryn, cyanazine
Senecio vulgaris		atrazine, simazine, prometryn, terbutryn, terbuthylazine, cyanazine, chloridazon, lenacil
Kochia scoparia	atrazine	simazine

one of the major mode-of-action groups in use (Devine & 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 & Eberlein 1997). A mutation to resistance at the position Pro_{197} was identified in *Lactuca serriola, Kochia scoparia, Brassica tournefortii, Sisymbrium orientale* and *Amaranthus retroflexus*; mutation at position Ala_{122} in *Xanthium strumarium, Amaranthus hybridus* and *Solanum ptycanthum*; mutation at position Ala_{205} in *Xanthium strumarium*; mutation at Trp_{574} in *Xanthium strumarium, Amaranthus hybridus, A. rudis, Kochia scoparia, Sisymbrium orientale* and *Ambrosia artemisiifolia*; and mutation at position Ser_{653} in *Amaranthus powellii, A. retroflexus* and *A. rudis* (Tranel & Wright 2002).

The resistance to ALS-inhibiting herbicides in seven Czech biotypes of *Kochia scoparia* is determined by a mutation at codon 574 of the ALS gene which induces a leucine for tryptophan substitution (Salava *et al.* 2004a).

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 aryloxyphenoxypropionates (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; Kobek *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 & Eberlein 1997; Devine & Shukla 2000). Monocots lack the prokaryotic form due to the loss of the *acc*D gene in the chloroplast genome. Multiple forms of eukaryotic ACCase are present in some grasses that differ in herbicide sensitivity (Konishi & Sasaki 1994; Devine & Shukla 2000). This is the primary basis for selectivity of these herbicides between grasses and dicots (Devine & Shukla 2000). Resistance of some grass species or cereal crops is based on an enhanced metabolic degradation of herbicides to inactive compounds (Devine & 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 & Powles 1993; Shukla *et al.* 1997a,b; Letouze & Gasquez 2000).

The alteration of the chloroplast multidomain-type ACCase is a frequent cause of resistance. It was concluded that a mutant allele of chloroplastic ACCase encoding a leucine residue instead of an isoleucine residue at position 1780 is a major gene of resistance to sethoxydim in *Setaria viridis*, *Lolium rigidum* and *Alopecurus myosuroides* (Délye *et al.* 2002a,b).

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 (e.g. Gronwald *et al.* 1989; Kemp *et al.* 1990; Preston & Powles 1997; De Prado *et al.* 1998; Devine & Preston 2000), see Table 4.

Among the enzymatic systems thought to be responsible for such resistance are glutathione-S-transferase 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).

In the following we list several examples of enzymes responsible for herbicide resistance due to an increased metabolism of herbicides.

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-trans-

Table 4. Weed species with herbicide resistance due to increased metabolism of herbicide

Weed species	Herbicides
Alopecurus myosuroides	Chlorotoluron
	Pendimethalin
	Diclofop-methyl
	Fenoxaprop-P-ethyl
	Propaquizafop
	Chlorsulfuron
Apera spica venti	Chlorotoluron
Abutilon theophrasti	Atrazine
Avena sterilis	Diclofop-methyl
Avena fatua	Triallate
Bromus tectorum	Chlorotoluron
Digitaria sanguinalis	Fluazifop-P-butyl
Echinochloa colona	Propanil
Echinochloa crus-galli	Propanil
Hordeum leporinum	Fluazifop-P-butyl
Lolium rigidum	Simazine
	Diclofop-methyl
	Fluazifop-P-butyl
	Tralkoxydim
	Chlorsulfuron
	Metribuzin
	Chlorotoluron
Phalaris minor	Isoproturon
Sorghum halepense	Bentazon
Stellaria media	Mecoprop

From Devine and Preston (2000)

ferases (Gronwald *et al.* 1989; 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 & 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 demethylations (Cole 1994; Devine & 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 cytP450-dependent detoxification (Coupland *et al.* 1990; Burnet *et al.* 1993; Maneechote *et al.* 1995; Carrera *et al.* 1999; Cocker *et al.* 1999; Singh *et al.* 1997).

Aryl acylamidase

Aryl acylamidases are soluble proteins of 32 kDA molecular weight that catalyse the removal of acylamides from generally hydrophobic substrates (Hoagland *et al.* 1974). The resistant *Echinochloa colona* populations have increased aryl acylamidase activity against a range of substrates, of which the most notable herbicide is propanil (Leah *et al.* 1994).

Resistance due to lack of herbicide activation

Triallate is actually a pro-herbicide, activated by a sulfoxidase to its active form (Devine & Shimabukuro 1994). The resistant *Avena fatua* biotype activates the herbicide at a rate of only one twelfth of that of the wild type. The lack of activation of the pro-herbicide within plants could lead to herbicide resistance (Kern *et al.* 1996). Triallate requires activation to the sulphone for activity (Casida *et al.* 1974), a reaction catalysed by a cytochrome P450 monooxygenase.

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 in-

herited cytoplasmically in most of the weed species (Jaseniuk *et al.* 1996). The gene conferring triazine resistance is located in the chloroplast genome (Holliday & Putwain 1980). The atrazine tolerance in *Abutilon theophrasti* was not cytoplasmically inherited (Andersen & 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).

Seeds of plants with special dispersal mechanisms can be spread a long distance from the mother plant or from the original site of the mother plant (e.g. by tumbling of *Salsola iberica* or *Kochia scoparia*) (Mallory-Smith *et al*. 1993; Saari *et al*. 1994).

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* 3000). 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 (Cifera *et al.* 1992). Some animals are responsible for spread of seeds nearby, while wind can move seeds over large distances (Darmency 1996).

FITNESS IN THE ABSENCE OF HERBICIDES

The probability of resistance occurring in a weed population prior to selection pressure by a herbicide will depend on the mutation frequency and size of the population, but overall on the relative fitness of the resistant genotypes, despite herbicide selection pressure (Gasquez 1997). When a resistant genotype is less fit than a susceptible one from the same population, the frequency of the resistant biotype decreases. If there is no difference in fitness or if the resistant biotype is even fitter, the latter genotype will remain dominant and never disappear (Gasquez 1997). The relative fitness of triazine resistant and susceptible biotypes in the absence of triazine has been reviewed by e.g. Holt (1988). The lack of fitness of most weeds resistant to triazine reduces the frequency at which biotypes can arise that have a cross- or multiple resistance to triazine and other types of herbicides. The triazine resistant genotypes are less competitive because the mutation reduces the electron transfer in the PS II (e.g. Conrad & Radosevich 1979; Chodová *et al.* 1990; Chodová & Mikulka 2002).

Lower ecological and physiological fitness was also reported in paraquat resistant biotypes of *Conyza canadensis* and *Hordeum glaucum* (Gressel 1991).

Results with other herbicides are to some extent ambiguous and it is not possible to generalize them.

CONCLUSIONS

For deciding on the direction of weed management strategy in future it is important to know how the herbicide resistance in weed populations evolved. Grass weeds (*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 (e.g. Matthews & Powles 1992; Preston *et al.* 1996; Heap 1997; 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 (e.g. 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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Souhrn

Chodová D., Mikulka J., Kočová M., Salava J. (2004): **Vznik, mechanismus a molekulární podstata rezistence plevelů vůči herbicidům**. Plant Protect. Sci., **40**: 151–168.

V práci jsou shrnuty literární poznatky a experimentální zkušenosti kolektivu autorů v oblasti výzkumu rezistence plevelů. Jsou popsány faktory, které podmiňují vznik rezistence. Chronologicky je uveden vznik rezistentních plevelů vůči devíti účinným látkám s odlišným mechanismem účinku a jejich rozšíření ve světě. Základním mechanismem rezistence je snížení citlivosti cílového místa účinku tzv. "target site resistance" a mechanismus spočívající v metabolické přeměně herbicidu na neúčinné látky. Jsou popsány funkce a genetické změny cílového místa účinku vybraných herbicidů. Jsou dokumentovány biotypy českých rezistentních plevelů s mutací v kodónu 264 *psb*A genu, který kóduje D1 protein a kodónu 574 genu acetolaktát syntázy.

Klíčová slova: rezistence vůči herbicidům; vznik a šíření rezistentních plevelů; cílové místo účinku; metabolická rezistence; molekulární základ rezistence

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