INVITED PAPER

Potential for Weeds to Develop Resistance to Sugarbeet Herbicides in North America

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ABSTRACT

Current knowledge on herbicide resistant weeds, mechanisms of herbicide resistance, fitness and adaptability, gene flow, and management of herbicide resistance was reviewed. Sixty eight percent of the 37 important problem weeds in sugarbeets have developed biotypes that are resistant to one or more herbicide classes in Africa, Australia, Europe, or North America. Eighteen of these weeds have biotypes that are resistant to herbicides in North America, but only two of these weeds have biotypes that are resistant to sugarbeet herbicides. Common lambsquarters is resistant to pyrazon in Switzerland and green foxtail is resistant to trifluralin in Canada. Diclofop methyl, fluazifop, paraquat, pyrazon, and trifluralin are considered high risk sugarbeet herbicides. Presently, it seems unlikely that problem weeds in sugarbeets in North America will develop resistance to sugarbeet herbicides or to other herbicides currently registered for use in sugarbeet rotations because sugarbeet growers integrate weed management programs, including crop and herbicide rotations and tillage. Preventive action against the evolution of herbicide- resistant weeds is advocated. Resistant weed biotypes in sugarbeets may increase as a result of the occurrence of cross- and multiple-resistance to many herbicides within the same biotype and the development of sugarbeet lines resistant to nonselective herbicides.

Additional Key Words: *Beta vulgaris L.*; crop rotations; plant breeding; weed resistant biotypes; weed management strategies.

Insect and pathogen resistance to pesticides has been a problem for more than 50 years for scientists and farmers attempting to control these pests. However, it was not until the mid-1980's that weed resistance to herbicides reached proportions that attracted scientist and farmer interest. By definition, herbicide resistant weeds (hereafter referred to as resistant weeds) survive and grow normally at the usually effective herbicide application rate (LeBaron and Gressel, 1982). The development of resistant weeds theoretically was possible anytime after the introduction of the first synthetic herbicide, 2,4-D[1], in 1945. However, it took 25 years before the first resistant weed was reported in North America (Bandeen et al., 1982). This weed species, common groundsel (*Senecio vulgaris* L.), became resistant to the triazine herbicide simazine.

Information on herbicide resistance in plants has been published in several excellent books and symposium proceedings (Caseley, 1990; Ford et al., 1987; Green et al., 1990; Holt, 1990; LeBaron, 1987a; and LeBaron and Gressel, 1982). In this article we review the current knowledge on resistant weeds, herbicide cross-resistant and multiple-resistant weeds, mechanisms of herbicide resistance, fitness and adaptability, gene flow, and management of herbicide resistance. This information is related to important problem weeds in sugarbeets that have biotypes that are resistant to non-sugarbeet or sugarbeet herbicides, and we discuss management strategies to prevent biotypes from developing resistance to sugarbeet herbicides and to herbicides used in rotational crops with sugarbeets.

HERBICIDE RESISTANT WEEDS

Resistance to pesticides is a global phenomenon that exists for fungicides, bactericides, insecticides, rodenticides, nematicides, and herbicides (Georghiou, 1986). Pests have demonstrated their ecological and biochemical adaptability to chemicals, some soon after they were first exposed. The selection of insects resistant to insecticides first was reported in 1908, of plant pathogens to fungicides in 1940, and of weeds to herbicides (*s*-triazines) in 1970 (LeBaron, 1982). However, as early as 1956, Harper predicted that in time weeds would become resistant to herbicides (Harper, 1956). The delay in the appearance of resistant weeds, relative to resistant insects and plant pathogens, is related to the slower generation time of plants, incomplete selection pressure of most herbicides, soil seed bank, plasticity of weedy plants, and apparent lower fitness of some resistant biotypes. All these factors could delay the evolution of resistance by keeping susceptible individuals in a population (Gressel and Segel, 1978).

[1]See Table 1 for common and trade names.

Table 1. Common and trade names of herbicides mentioned in this review and risk level associated with the selection of herbicide resistant weeds.

Common name	Trade name	Herbicide class	Herbicide resistance weed risk level[†]
Atrazine	AAtrex	Triazines	High
Barban	Carbyne	Carbamates	Low
Bromoxynil	Brominal, Buctril	Uracils	Low
Clopyralid	Stinger	Pyridinoxy acids	Low
Cyanazine	Bladex	Triazines	High
Cycloate	RoNeet	Thiocarbamates	Low
Desmedipham	Betanex	Carbamates	Low
Desmedipham + phenmedipham	Betamix	Carbamates	Low
Diclofop methyl	Hoe-Grass Hoelon	Diphenyl ethers	High
Diethatyl	Antor	Amides	Low
Diuron	Karmex	Ureas	High
Endothall	Herbicide 273	Phthalic acids	Low
Ethofumesate	Nortron	Unclassified	Low
EPTC	Eptam, Genap	Thiocarbamates	Low
Fluazifop	Fusilade	Diphenyl ethers	High
Glyphosate	Roundup	Unclassified	Low
Imazethabenz methyl	Assert	Imidazolinones	High
Imazethapyr	Pursuit Event	Imidazolinones	High
Lenacil	Venzar	Uracils	High
Linuron	Lorox	Ureas	High
MCPA	several	Phenoxys	Low
Metamitron	Goltix	Triazines	High
Metsulfuron methyl	Ally	Sulfonylureas	High
Nicosulfuron	Accent	Sulfonylureas	High

Table 1. (continued)

Common name	Trade name	Herbicide class	Herbicide resistance weed risk level[†]
Paraquat	Paraquat	Bipyridiliums	High
Pebulate	Tillam	Thiocarbamates	Low
Pendimethalin	Prowl	Dinitroanilines	High
Pyrazon	Pyramin	Diazinones	High
Primisulfuron	Beacon	Sulfonylureas	High
Sethoxydim	Poast	Cyclohexones	Low
Simazine	Princep	Triazines	High
TCA	Sodium TCA	Aliphatics	Low
Triallate	Avadex BW	Thiocarbamates	Low
Tribenuron methyl	Express	Sulfonylureas	High
Tribenuron methyl + thifensulfuron	Harmony Extra	Sulfonylureas	High
Trifluralin	Treflan	Dinitroanilines	High
2,4-D	several	Phenoxys	Low

[†]LeBaron and McFarland (1990).

The number of weed species biotypes that have developed resistance to herbicides continues to increase since the first report of triazine resistance in 1970 for common groundsel in Washington (Ryan, 1970). In 1986, there were 49 weed species documented with biotypes resistant to triazines, and 9 species with biotypes resistant to other herbicide classes (LeBaron, 1987b). By 1990, 107 resistant weed biotypes were documented throughout the world (LeBaron, 1990). Fifty percent of these biotypes are resistant to triazine herbicides and the remainder to the other 14 herbicide classes. Resistant biotypes have been reported in all but 10 states and 2 provinces of Canada. Unless weeds are managed properly over time, resistant biotypes can dominate the population and the soil seed bank. Resistance in the field becomes visible or detectable when it reaches about 30% of the population (Gessel and Segel, 1978). In most instances with the triazines, resistance appeared after seven or more years of repeated use (Bandeen et al., 1982). However, resistance to the sulfonylureas has appeared after only three to five years of use (Mallory- Smith et al., 1990).

HERBICIDE CROSS-RESISTANT WEEDS

Herbicide cross-resistant weeds are biotypes that have developed resistance after selection by one herbicide, then subsequently exhibit resistance to herbicides that differ chemically and have different modes of action (Powles and Howat, 1990). A biotype of rigid ryegrass (*Lolium rigidum* Gaud.) was the first weed reported to exhibit extensive cross-resistance (Heap and Knight, 1986). Biotypes of rigid ryegrass have developed cross-resistance to cyclohexone, dinitroaniline, diphenyl ether, sulfonylurea, and triazine herbicides (Powles and Howat, 1990). At least six other weed species have biotypes that exhibit cross-resistance (Mallory-Smith et al., 1990; Moss and Cussans, 1987; Primiani et al., 1990; Thill et al., 1990; Vaughn et al., 1990). Three of these weed species, common chickweed[2], kochia, and Russian thistle, are weeds in sugarbeets (Schweizer and Dexter, 1987). Biotypes of common chickweed and Russian thistle have developed cross-resistance to sulfonylurea herbicides (Thill, et al., 1990). A biotype of kochia has developed cross-resistance to sulfonylurea (Saari et al., 1990; Thill et al., 1990) and imidazolinone herbicides (Primiani et al., 1990). The kochia biotype was resistant to herbicides with the same mode of action, e.g., sulfonylureas and imidazolinones, but this biotype was not resistant to herbicides with different modes of action, e.g., atrazine, bromoxynil, MCPA, and diuron. Cross-resistance no doubt will become evident in other weed species.

MULTIPLE HERBICIDE RESISTANCE

Multiple herbicide resistance is a relatively new phenomenon in which plant populations display resistance to a series of chemically unrelated pesticides having different modes of action (Gressel, 1988). Multiple- resistance may occur as a linked or pleiotropic trait or as secondarily evolved resistance (LeBaron and McFarland, 1990). Weeds that exhibit cross tolerance to herbicides within the same family, or to herbicides with a similar mode of action, easily can be understood. More disturbing, however, are the few documented cases of weeds that are resistant to structurally unrelated herbicides having totally different modes of action. This multiple herbicide resistance generally is thought to be due to enhanced plant detoxification of xenobiotics, probably through elevated monooxygenase enzyme levels in resistant plants (Holtum et al., 1989). Such detoxification activities of mixed function oxidases (MFO's) in mammals, insects, fungi, and plants are well documented (Hatzios and Penner, 1982; Komives and Dutka, 1989). These enzymes have little substrate specificity, are inhibited by carbon dioxide and piperonyl butoxide, require

[2]See Tables 3 and 4 for scientific names of weed species.

oxygen and NADPH to function, and require the involvement of an electron transport system. MFO's likely represent metabolic herbicide resistance in some weeds, compared to altered site of action resistance more commonly encountered in resistant weeds.

MECHANISMS OF HERBICIDE RESISTANCE

Herbicide resistance in weeds is due to altered sites of action, enhanced metabolism, or sequestering. Most commonly, it is due to an altered site of action. This is true for weeds that are resistant to dinitroaniline, sulfonylurea, and triazine herbicides. The primary site of action for triazines is the inhibition of photosystem II in chloroplasts (Arntzen et al., 1982), and it is the inhibition of acetolactate synthase (ALS) for imidazolinone (Shaner et al., 1984) and sulfonylurea herbicides (Primiani et al., 1990). Dinitroaniline herbicides, such as trifluralin, disrupt mitosis by interfering with tubulin formation (Vaughn et al., 1990). However, in some velvetleaf biotypes, resistance to atrazine results from enhanced metabolism and not from an altered site of action (Gronwald et al, 1989). Resistance to paraquat appears to be due to a mechanism that sequesters paraquat from its site of action in the chloroplast (Fuerst and Vaughn, 1990). In sulfonylurea resistant biotypes studied to date, resistance is due to an altered site of action. The activity of the ALS enzyme in resistant biotypes is inhibited much less by sulfonylurea herbicides than in susceptible biotypes (Saari et al., 1990; Thill et al., 1990). For ALS resistance, the proposed mechanism of action is an altered amino acid sequence in the ALS enzyme, which then loses its affinity for ALS-inhibiting herbicides (Hartnett et al., 1990; Saari et al., 1990).

FITNESS

Fitness describes the evolutionary advantage of a phenotype, based on its survival and reproductive success (Crow, 1986). Relative fitness of susceptible (S) and resistant (R) weed biotypes has important consequences for management of herbicide resistance. R biotypes can be less fit or just as fit as S biotypes. When a R biotype is less fit than a S biotype, discontinuous use of the selective herbicide allows natural selection to restore the predominance of susceptible species. This often has been the case with the triazine herbicides. With other herbicide classes, R biotypes of several weed species have been reported to be as fit as the S biotypes (Heap and Knight, 1986; Mudge et al., 1984). Consequently, if the fitness of a R biotype is the same as a S biotype, resistance may decline slowly, if at all, and very different tactics for managing resistance may be necessary to restore susceptibility (Gressel, 1987; Gressel and Segel, 1990).

FITNESS AND ECOLOGICAL ADAPTABILITY OF HERBICIDE RESISTANT BIOTYPES

Natural selection for a particular trait, such as herbicide resistance, may incur a cost to the organism in terms of fitness, or its ability to survive and reproduce. This has been found for biotypes possessing the maternally inherited trait of triazine resistance (Maxwell et al., 1990). For most triazine resistant biotypes reported to date, the mechanism of resistance is a single amino acid modification in a 32 kD thylakoid protein, resulting in reduced binding of triazine herbicides (Marx, 1983). This mutation has a detrimental effect on photosynthesis, resulting in decreased biomass production and seed production. However, compensatory interactions of the chloroplast and nuclear genes may partially overcome reduced productivity. Expression of reduced productivity also appears to be regulated or influenced by environmental conditions (Holt, 1990). Whether similar trends in relative fitness will be found in weeds resistant to ALS-inhibiting herbicides remains to be examined.

GENE FLOW

Gene flow describes the processes that influence the maintenance of a particular genotype in a population (Maxwell et al., 1990). Genes immigrate into plant populations via pollen and seed, and this immigration can be predicted with models (Maxwell et al., 1990). Model simulations suggest that gene flow can significantly decrease the proportion of resistance in cross-pollinating species (outcrossers), but gene flow alone probably will not effectively reduce the proportion of resistance in predominately self- fertilizing populations (Roush et al., 1990).

The relative fitness of weed biotypes in mixed populations is heavily influenced by fecundity, survivorship, and plant competition (Maxwell et al., 1990). Where herbicide resistance is coded by a single gene, use of the Hardy-Weinberg model may predict the level of a given biotype in a mixed population (Crow, 1986; Gwynne and Murray, 1985). However, use of a persistent, highly selective herbicide may dramatically shift the population equilibrium in favor of less fit, resistant biotypes, and may cause the rapid increase of resistant plant numbers which generally are first noticed when they represent 30% or more of the total population. Upon removal of the herbicide selection pressure, reversion to a more susceptible population may be slow, since population dynamics will then be governed by all the processes that contribute to the fitness of each biotype (Maxwell et al., 1990). Susceptible biotypes will, however, eventually return to Hardy-Weinberg equilibrium when herbicides are removed from the system.

MANAGEMENT OF HERBICIDE RESISTANCE

The key to avoiding resistant weed problems is the use of management techniques to prevent problems from developing. Practices that can help prevent the development of resistant weeds include crop rotation, herbicide rotation, and choice of tillage methods. With crop rotation, weeds resistant to herbicides in one crop often are controlled with herbicides used in alternative crops. With herbicide rotation, herbicides in different chemical classes, and with different mechanisms of action, should be used to avoid the problems of cross- or multiple-resistance (Holt and LeBaron, 1990). Weeds often can be controlled better throughout the growing season with conventional tillage (moldboard plowing and discing) as compared to minimum tillage (chisel plowing and discing) or no-till (Ritter et al., 1985).

The underlying principle of any management strategy is to reduce the selection pressure for the evolution of resistance. The ability to predict which herbicides will have a greater chance of selecting for resistance would be useful. LeBaron and McFarland (1990) proposed several characteristics of herbicides and their use that contribute to a high probability for the evolution of herbicide resistance: a) single target site and specific mode of action, b) extremely active and effective in killing all genotypes within a single species, c) long soil residual and season-long control of germinating weeds, d) applied frequently and over several growing seasons without rotating, alternating, or combining with other herbicide classes, and e) management practices that likely reduce the genetic variance in weed species. These characteristics would cause intense selection pressure favorable for the evolution of resistance. Herbicide classes that meet some or all of these criteria and are at higher risk would be the bipyridiliums, diazinones, dinitroanilines, diphenyl ethers, imidazolinones, sulfonylureas, triazines, uracils, ureas, and some unclassified herbicides. Such herbicides would more likely select for resistance than would those that meet few or none of the criteria. Only five registered sugarbeet herbicides are represented in the high risk group: Fusilade, Hoe-Grass (Hoelon), Paraquat, Pyramin, and Treflan (Table 2). Herbicide classes that meet fewer of these criteria and are at lower risk would be the aliphatics, amides, benzoics, carbamates, cyclohexones, nitriles, organic arsenicals, phenoxys, phthalic acids, pyridinoxy acids, thiocarbamates, and some unclassified herbicides. The majority of registered sugarbeet herbicides, fortunately, are represented in the low risk group (Table 2).

Several prediction models have been developed to evaluate management tactics that prevent, delay, or reduce resistance. Gressel and Segel's (1990) models suggest that the best tactics to prevent or delay the appearance of R populations are: a) use

Table 2. The risk of developing herbicide resistant weeds for sugarbeet herbicides registered for use in North America.

	Risk of development of herbicide† resistant weeds	
Herbicide class	High	Low
Aliphatics	—	TCA
Amides	—	ANTOR
Bipyridiliums	PARAQUAT	—
Carbamates	—	BETAMIX
	—	BETANEX
	—	CARBYNE
Cyclohexones	—	POAST
Diazinones	PYRAMIN	—
Dinitroanilines	TREFLAN	—
Diphenyl ethers	FUSILADE	—
	HOE-GRASS, HOELON	—
Phthalic acids	—	HERBICIDE 273
Pyridinoxy acids	—	STINGER
Thiocarbamates	—	AVADEX BW
	—	EPTAM
	—	RONEET
	—	TILLAM
Unclassified	—	NORTRON
	—	ROUNDUP

†See Table 1 for herbicide common names and classes.

herbicide treatments with the minimum selection pressure that gives cost-effective weed control, but leaves enough S seeds each year to dilute out R seeds; b) use herbicide mixtures or use rotations of herbicides that act at different sites and have different modes of degradation; and, c) employ mechanical cultivation in the rotation, especially if it preferentially controls unfit R biotypes.

A recent model simulates the evolution, spread, and subsequent dynamics of resistance in the presence and absence of a herbicide (Maxwell et al., 1990). The model identified two sets of biological processes as key factors in the evolution and dynamics of R weed populations—ecological fitness and gene flow. The following management tactics and/or implications were suggested: a) reducing herbicide efficacy by intentionally

leaving skips during herbicide application provides enough healthy S individuals in the population to reduce the levels of resistance through fitness and gene flow processes; b) leaving untreated adjacent rows or maintaining S populations of the weeds dispersed through the treated population increases the potential for immigrating S weed pollen and seed to decrease the role of evolution; c) the most significant influence of relative competitive ability on resistance dynamics occurs in the recovery period following suspension of herbicide use when crop density was increased under the assumption that the R phenotype is less competitive with the crop than the S phenoptype; and d) the potential for managing resistance is improved by creating a source of the S weed phenotype to augment the effect of a new herbicide that will control both R and S weed phenotypes.

IMPORTANT PROBLEM WEEDS IN SUGARBEETS IN NORTH AMERICA

Unlike insects, diseases, and nematodes, weeds occur in all sugarbeet fields every year at population levels that will cause crop failure unless they are controlled (Jansen, 1972). Over 250 plant species are considered important weeds throughout the world. Two perennial weeds, quackgrass and field bindweed, and eight annual weeds comprise the list of major weeds in sugarbeet production areas in both North America and the world (Holm et al., 1977). The annual dicots are common chickweed, common lambsquarters, prostrate knotweed, redroot pigweed, wild buckwheat, and wild mustard; the monocots are barnyardgrass and green foxtail. No resistant biotypes of field bindweed, prostrate knotweed, and quackgrass have been reported in the world.

RESISTANT BIOTYPES OF IMPORTANT PROBLEM WEEDS IN SUGARBEETS IN NORTH AMERICA

Thirty seven important problem weeds are present in sugarbeet production regions in North America (Schweizer and Dexter, 1987). The important problem weeds in sugarbeets in North America are comprised of 29 dicots (Table 3) and 8 monocots (Table 4). Twenty five of these weed species have biotypes that are resistant to herbicides in one or more countries. Eighteen of these weed species have biotypes that are resistant to herbicides in North America (Figure 1). Six of these weed species have biotypes that are resistant only in Europe (Figure 1 and Table 3), and one species, wild oats, has biotypes that are resistant in Africa and Australia (Table 4). Thus, 68% of the important problem weeds in sugarbeets in North America already have developed biotypes that are resistant to one or more herbicide classes in Africa, Australia, Europe, or North America.

Table 3. Resistant and non-resistant biotypes† of important problem dicot weed species in sugarbeets in North America by herbicide class and geographic location.

Common name	Genus and species	Herbicide class	Geographic location
Dicots having resistant biotypes			
Black nightshade	*Solanum nigrum* L	Triazines	USA, Europe
Canada thistle	*Cirsium arvense* (L.) Scop.	Phenoxys	Europe
Common chickweed	*Stellaria media* (L.) Vill.	Phenoxys	Europe
		Imidazolinones	Canada
		Sulfonylureas	Canada
		Triazines	Europe
Common cocklebur	*Xanthium strumarium* L.	Arsenicals	USA
Common lambsquarters	*Chenopodium album* L.	Nitriles	Europe
		Ureas	Europe
		Diazinones	Europe
		Triazines	USA, Canada
			Europe
Common ragweed	*Ambrosia artemisiifolia* L.	Triazines	USA, Canada
			Europe
Kochia	*Kochia scoparia* (L.) Schrad.	Imidazolinones	USA,Canada
		Sulfonylureas	USA,Canada
		Triazines	USA
Ladysthumb	*Polygonum persicaria* L.	Triazines	Europe
Longleaf groundcherry	*Physalis longifolia* Nutt.	Triazines	USA
Pale smartweed	*Polygonum lapathifolium* L.	Triazines	Europe
Powell amaranth	*Amaranthus powellii* S.Wats.	Triazines	USA, Canada
Prostrate knotweed	*Polygonum aviculare* L.	Triazines	Europe
Prostrate pigweed	*Amaranthus blitoides* S. Wats.	Triazines	Europe
Redroot pigweed	*Amaranthus retroflexus* L.	Carbamates	Europe
		Ureas	Europe
		Uracils	Europe
		Triazines	USA, Canada
			Europe
Russian thistle	*Salsola iberica* Sennen & Pau.	Imidazolinones	USA
		Sulfonylureas	USA
Shepherdspurse	*Capsella bursa-pastoris* (L.) Medik.	Triazines	Europe

Table 3. (continued)

Common name	Genus and species	Herbicide class	Geographic location
Smooth pigweed	*Amaranthus hybridus* L.	Carbamates	Europe
		Ureas	Europe
		Uracils	Europe
		Triazines	USA, Europe
Velvetleaf	*Abutilon theophrasti* Medik.	Triazines	USA
Wild buckwheat	*Polygonum convolvulus* L.	Triazines	USA, Europe
Wild mustard	*Sinapis arvensis* L.	Triazines	Canada
Dicots having no confirmed resistant biotypes			
Burning nettle	*Urtica urens* L.		
Common mallow	*Malva neglecta* Wallr.		
Common purslane	*Portulaca oleracea* L.		
Common sunflower	*Helianthus annuus* L.		
Field bindweed	*Convolvulus arvensis* L.		
Hairy nightshade	*Solanum sarrachoides* Sendtner		
Jimsonweed	*Datura stramonium* L.		
Little mallow	*Malva parviflora* L.		
Perennial sowthistle	*Sonchus arvensis* L.		

†From LeBaron, 1990.

Table 4. Resistant and non-resistant biotypes† of important problem monocot weed species in sugarbeets in North America by herbicide class and geographic location.

Common name	Genus and species	Herbicide class	Geographic location
Monocots having resistant biotypes			
Barnyardgrass	*Echinochloa crus-galli* (L.)Beauv.	Amides	Europe
		Triazines	USA, Canada
			Europe
Green foxtail	*Setaria viridis* (L.) Beauv.	Dinitroanilines	Canada
		Triazines	Europe
Italian ryegrass	*Lolium multiflorum* Lam.	Diphenyl ethers	USA
Yellow foxtail	*Setaria glauca* (L.) Beauv.	Triazines	USA, Canada
			Europe
Wild oats	*Avena fatua* L.	Diphenyl ethers	Africa
			Australia
Monocots having no confirmed resistant biotypes			
Johnsongrass	*Sorghum halepense* (L.)Pers.		
Quackgrass	*Agropyron repens* (L.) Beauv.		
Yellow nutsedge	*Cyperus esculentus* L.		

†From LeBaron, 1990.

Conceivably, the other 12 important weed species in sugarbeets in North America may eventually develop R biotypes. Nine of these species are dicots: burning nettle, common mallow, common purslane, common sunflower, field bindweed, hairy nightshade, jimsonweed, little mallow, and perennial sowthistle (Table 3). The other three species are monocots: johnsongrass, quackgrass, and yellow nutsedge (Table 4). The R biotypes of important problem dicot weed species have developed resistance to ten herbicide classes: arsenicals, carbamates, diazinones, imidazolinones, nitriles, phenoxys, sulfonylureas, triazines, uracils, and ureas (Table 3). The resistant biotypes of important problem monocot weed species have developed resistance to four herbicide classes: amides, dinitroanilines, diphenyl ethers, and triazines (Table 4).

Of the 14 herbicide classes enumerated in Tables 3 and 4, sugarbeet herbicides are represented in only 5: amides, carbamates, diazinones, dinitroanilines, and diphenyl ethers (Table 2). Diazinone, dinitroaniline, and diphenyl ethers classes are considered high risk and would more likely lead to the selection of R biotypes (LeBaron and McFarland, 1990). Registered sugarbeet herbicides in the high risk group are Fusilade, Hoe-Grass (Hoelon), Paraquat, Pyramin, and Treflan.

WEEDS RESISTANT TO SUGARBEET HERBICIDES

Based on past history and the presently increasing problems and consequences from insect and disease resistance to pesticides, and weed resistance to 15 herbicide classes (LeBaron, 1990), weed scientists and agriculturalists are concerned about the development of weed resistance to sugarbeet herbicides. Yet only two important problem sugarbeet weeds, common lambsquarters and green foxtail, have developed resistance. A biotype of common lambsquarters from Switzerland is resistant to pyrazon and partially resistant to metamitron (LeBaron and Gressel, 1982), and another biotype from Hungary is resistant to lenacil (Mikulka, 1988). A biotype of green foxtail from Canada is resistant to trifluralin (LeBaron and McFarland, 1990). Only pyrazon and trifluralin are registered for use in the crop in North America. Despite 35 years of increasingly intensive herbicide usage in the United Kingdom, weed resistance has not developed in sugarbeet fields (Gwynne and Murray, 1985).

There are several hypotheses explaining this lack of weed resistance. One explanation suggests that biotypes resistant to sugarbeet herbicides make up an infinitesimally small proportion of the "natural" population, and these biotypes are ecologically "less fit," as proposed by Gressel and Segel (1978), than the sensitive biotypes. Thus, resistant biotypes will not be detected unless the repeated use of sugarbeet herbicides occurs. Others feel that weed resistance to sugarbeet herbicides has

been minimal because growers employ management programs of integrated weed control, including conventional tillage, inter-row cultivation, and crop and herbicide rotations. Currently, we do not know the answers. It is clear, however, that *s*-triazine resistance can be overcome, but the control of triazine resistant weeds may be more costly with other weed management strategies. We also know that some genera that contain herbicide-resistant and herbicide-tolerant populations, e.g., *Amaranthus, Chenopodium,* and *Polygonum,* provide major weeds in many sugarbeet fields.

SELECTION OF HERBICIDE RESISTANT WEED BIOTYPES IN SUGARBEET ROTATIONS

As pointed out above, one of the key management techniques to prevent development of R weed biotypes is to rotate herbicides in crop rotations. A typical crop rotation in the Central Great Plains is barley-corn-pinto bean- sugarbeet. How important is it, then, to rotate herbicides in this rotation to avoid resistant biotypes of any of the 37 important problem weed species in sugarbeets in North America? To address this question, we reviewed all of the registered herbicides for each crop by weed species, and listed those herbicides by crop and weed species where R weed biotypes for each herbicide were documented and where the selection pressure for the evolution of resistance was considered a high risk based on the criteria discussed above. This information is summarized in Table 5.

The probability of selecting for R biotypes with any of the important problem weed species in sugarbeets in North America is very low with a barley- corn-pinto bean-sugarbeet rotation in the Central Great Plains. The only instance might be with green foxtail, if Treflan were used in each crop each year (Table 5). The likelihood of this is remote, because this herbicide is seldom used in barley and corn.

With the other weed species, selection for R biotypes of kochia and Russian thistle could occur for two to three consecutive years with the use of several high risk herbicides in barley, corn, and pinto beans (Table 5). However, it is unlikely that R biotypes of these two weeds would appear because in most instances it has taken three or more years of repeated herbicide use before R biotypes appeared in the field.

Of special concern in the future is the occurrence of multiple- or cross-resistance to many herbicides within the same biotype, apparently by metabolic detoxification, and the development of sugarbeet cultivars resistant to nonselective herbicides. A sugarbeet line has been developed that is resistant to a number of sulfonylurea herbicides (Saunders et al., 1990). Excitement is high about the potential benefits of sulfonylurea-resistant sugarbeet lines because of increased rotational

Table 5. These high risk herbicides† when used in barley-corn-pinto bean-sugarbeet rotation in the U. S. Central Great Plains have the potential to select for herbicide resistant weeds in the rotation.

Common name	Herbicide class	Barley	Corn	Pinto beans	Sugar-beets
Dicots having resistant biotypes in North America					
Black nightshade	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Common chickweed	Sulfonylureas	ALLY	—	—	—
		HARMONY EXTRA	—	—	—
		EXPRESS	—	—	—
Common cocklebur	Arsenicals	—	—	—	—
Common lambsquarters	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Common ragweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Groundcherries	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Kochia	Imidazolinones	ASSERT	—	PURSUIT	—
	Sulfonylureas	ALLY	BEACON	—	—
		HARMONY EXTRA	—	—	
		EXPRESS	—	—	—
	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Powell amaranth	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Redroot pigweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Russian thistle	Imidazolinones	ASSERT	—	PURSUIT	—
	Sulfonylureas	ALLY	BEACON	—	—
		HARMONY EXTRA	—	—	—
		EXPRESS	—	—	—
Smooth pigweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Velvetleaf	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Wild buckwheat	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—

Table 5. (continued)

Common name	Herbicide class	Barley	Corn	Pinto beans	Sugar-beets
Wild mustard	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Monocots having resistant biotypes in North America					
Barnyardgrass	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Green foxtail	Dinitroanilines	—	PROWL	PROWL	—
		—	—	SONALAN	—
		TREFLAN	TREFLAN	TREFLAN	TREFLAN
Italian ryegrass	Diphenyl ethers	HOE-GRASS	—	—	—
Yellow foxtail	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Dicots having resistant biotypes in European countries					
Common lambsquarters	Nitriles	BUCTRIL	BUCTRIL	—	—
	Diazinones	—	—	—	PYRAMIN
	Ureas	—	LOROX	—	—
Ladysthumb	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Pale smartweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Prostrate knotweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Prostrate pigweed	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
	Ureas	—	LOROX	—	—
Shepherdspurse	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Monocots having resistant biotypes in Africa, Australia, and Europe					
Barnyardgrass[‡]	Amides	—	—	—	—
Green foxtail[‡]	Triazines	—	ATRAZINE	—	—
		—	BLADEX	—	—
Wild oats[§]	Diphenyl ethers	HOE-GRASS	—	—	—

[†]See Table 1 for common names of herbicides.
[‡]Resistant biotypes in Europe.
[§]Resistant biotypes in Africa and Austrailia.

flexibility, broader spectrum weed control, and the short soil residual of some sulfonylurea herbicides. The risk is that an increasing number of weeds have developed resistance to sulfonylureas and other ALS inhibitors (LeBaron, 1990). Chickweed, kochia, and Russian thistle are three important problem dicot weed species in sugarbeets in North America that have developed resistance to sulfonylureas (Table 3). Thus, the industry should not develop and market ALS resistant crops or crops resistant to only one herbicide with a high risk for resistance for the purpose of greatly expanding their use (LeBaron and McFarland, 1990). Herbicide-resistant crops should be developed for minor crops to provide more flexibility in control of resistant weeds, to enhance herbicide selectivity in crop varieties, and to avoid crop injury from long soil residuals (LeBaron and McFarland, 1990).

Development of herbicide resistant weeds in crops, including sugarbeets, could be accelerated by marketing new high risk herbicides or use of biotechnology to engineer crops resistant to these herbicides. Plant breeders and geneticists should devote more time to developing sugarbeet genotypes that tolerate nonselective herbicides with a low risk for weed resistance. In vitro selection techniques have been developed to identify herbicide tolerant genotypes within heterogeneous seedling populations (Saunders et al., 1990; Smith and Moser, 1985). Genotypes that tolerate Nortron, a low risk sugarbeet herbicide (Table 2), have been identified through in vitro selection techniques (Smith and Moser, 1985).

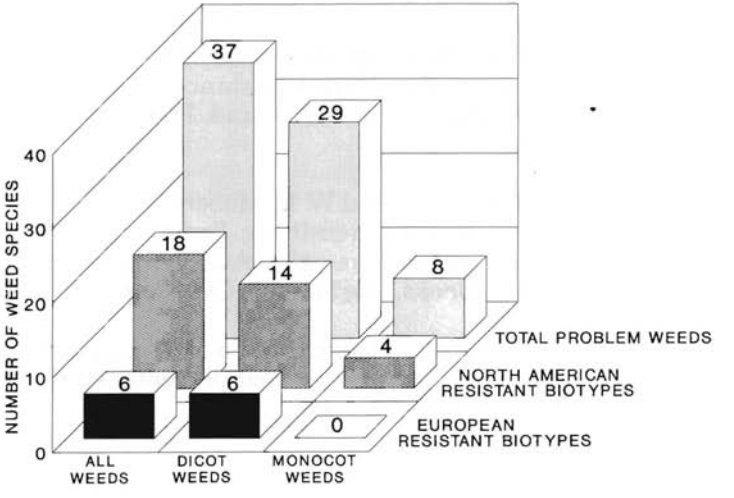

Figure 1. Numbers of important problem dicot and monocot weed species in sugarbeets in North America and proportion of those that have developed herbicide resistance in Europe or North America.

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