

# History, Mechanisms, and Strategies for Prevention and Management of Herbicide Resistant Weeds<sup>1</sup>

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Herbicide resistance is the inherited ability of a plant to survive and reproduce following exposure to a dose of herbicide normally lethal to the wild type. In a plant, resistance may be naturally occurring or induced by such techniques as genetic engineering. Resistance may occur in plants by random and infrequent mutations; no evidence has been presented to demonstrate herbicide-induced mutation. Through selection, where the herbicide is the selection pressure, susceptible plants are killed while herbicide resistant plants survive to reproduce without competition from susceptible plants. If the herbicide is continually used, resistant plants successfully reproduce and become dominant in the population. Thus, the appearance of herbicide resistance in the population is an example of rapid weed evolution.

## History

Herbicide resistance was first reported in 1957 (Hilton 1957, Switzer 1957). In California, common groundsel (*Senecio vulgaris*) was the first reported herbicide resistant weed species (Holt 1988). It was shown to be resistant to herbicides in the triazine chemical class. Since that time, plants of 61 species (42 dicots and 19 monocots) have evolved resistance to the triazine herbicides. Herbicide resistance in plants did not evolve as early as insecticide or fungicide resistance due to fundamental life cycle and genetic differences between plants, insects, and fungi. The delayed appearance of resistant weeds relative to insects and fungi is generally attributed to slower generation time of plants, incomplete selection pressure of most herbicides, soil seed reserve, and plasticity of weedy plants, all of which keep susceptible individuals in a population and thus delay evolution of resistance (Holt 1992). The appearance of herbicide resistance in plants is currently increasing at an exponential rate (Figure 1), mirroring the trends previously seen with insecticide and fungicide resistance. Besides triazine resistance, there are biotypes of over 150 weed species expressing resistance to 14 other herbicide classes. The most common mechanism of action or target site of herbicides, the chemical class, and the number of species with biotypes resistant to each herbicide class are summarized in Table 1.

In California, herbicide resistance currently is most widespread in aquatic weeds in rice production (Table 2). Many of these weed species have been selected for resistance to the sulfonylurea herbicide bensulfuron (Anonymous 1993). In addition, there has been one report of

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triazine resistance as well as two reports of sulfonylurea resistance in a non-crop area. A roadside survey conducted in 1995 and 1996 found that resistance to sulfonylurea herbicides was common in Russian thistle (*Salsola tragus*). Most recently, a rigid ryegrass (*Lolium rigidum*) biotype exhibited resistance to glyphosate in a northern California orchard (Heap 1999; Dave Bayer, pers. comm.). Despite these examples, there are fewer reports of resistance in California to date than in other regions of the United States (Table 1). However, current and pending registrations in California primarily involve herbicides that act on amino acid synthesis (Accent, Pursuit, Shade-Out, and Upbeet). Use of herbicides in this group has selected resistance in many weed species. Since these herbicides lead to rapid selection for resistant weeds, the number of cases in California has increased and this trend is expected to continue. In addition, a number of genetically engineered crops that are resistant to specific herbicides will soon be available in California such as Roundup Ready cotton and corn. Sole reliance on the specific herbicide used in these resistant crop varieties will increase the selection pressure for resistance to that herbicide. Herbicide-resistant crops will not be an end-all solution to weed problems and they will lose their effectiveness for weed management if used continuously.

## Mechanisms

Evolution and natural selection are the processes that have led to the plant species found around the world today. Many plants, particularly weeds, contain a tremendous amount of genetic variation that allows them to survive under a variety of environmental conditions. Most herbicides act on a single specific site of action. This site of action is usually under the control of a single gene, or at most a few genes. With a single gene mutation, even minor changes in gene expression can confer resistance by modifying the site where the herbicide has its toxic effect (site of action). The evolution of a resistant population of a species is brought about through selection pressure imposed by that herbicide or class of herbicides. When a herbicide exerts selection pressure on a population, plants possessing the resistance trait have a distinct advantage. Unlike the susceptible plants in this population, resistant individuals will survive and reproduce. Continuous herbicide exposure maintains the selection pressure, thereby rapidly increasing the number of resistant plants.

Some weeds have traits that promote the evolution of resistance. High seed production with most seed germinating within a year can accelerate the evolution of resistance. This occurs because susceptible plants are removed rapidly from the population by the herbicide, thus increasing the proportion of individuals possessing the trait that confers resistance. High seed production coupled with genetic variation increases the probability of resistance evolution. Perennial weeds, particularly those with vegetative reproductive tissues, are less likely to evolve resistance compared to weeds with an annual life cycle that produce abundant seeds since there is less genetic diversity in the population and less reliance on seed production, which would perpetuate resistance.

In the absence of herbicide treatment, weeds with resistance to the triazine herbicides are not as fit as susceptible plants of the same species. This is due to a reduction in the efficiency of photosynthesis in resistant plants caused by an alteration in a specific photosynthetic protein that is also the herbicide binding site, which confers resistance. Since resistant plants are less fit, they reproduce at lower rates and, consequently, represent a smaller fraction of the number of

individuals within a population. In contrast, some resistance traits do not incur the same fitness cost and, thus, often represent a larger fraction of individuals within a population. The frequency of the resistance trait within the population is an important factor in the rate of selection for resistance among weed species. For example, resistance to triazines took 10 years of continual use to evolve. Unlike the triazines, the sulfonylurea herbicides (inhibitors of ALS, acetolactate synthase) have no fitness cost associated with the resistance trait. Resistance to these herbicides took only 4 years to evolve. For weed species with resistance to sulfonylurea herbicides, it has been estimated that the initial proportion of resistant plants in a population is approximately one-in-a-million individuals. Thus, if a weed population has a density of 10 plants per m<sup>2</sup>, one resistant individual would be expected for every 10 hectares (24.7 acres) of infestation. Without multiple control strategies, the resistant individual likely will survive to produce resistant seed.

There are several factors such as herbicide characteristics, plant characteristics, weed control practices, and production practices that increase the probability of selection for herbicide resistance. Herbicide factors that contribute to the potential for resistance include long soil residual activity, single target site and specific mode of action, and high effective kill of a wide range of weed species. Herbicides with prolonged soil residual activity have a longer time to select for the resistance trait since they will kill most susceptible plants that germinate over a growing season. Herbicides with a single target site controlled by few genes are more likely to encounter plants with mutations for resistance than are herbicides with several modes of action. High effective kill rapidly depletes susceptible genes from the population, resulting in a rapid increase in resistance from few initial plants.

Although the most common mechanism of herbicide resistance in weeds is an alteration at the site of action, resistance can also result from an enhanced ability of the plant to metabolize and detoxify the herbicide (Holt et al. 1993). This latter mechanism, however, is not yet widespread in the United States. Like target site changes, selection for enhanced metabolism can also occur with repeated application of the same herbicide or with herbicides that are affected by the same detoxification enzymes. For example, enhanced metabolism is thought to confer resistance to picolinic acid herbicides in yellow starthistle in eastern Washington. Weed biotypes with enhanced metabolism have a much lower level of resistance compared to weeds expressing resistance through site of action changes. Selection for weeds with enhanced metabolism is more rapid when a herbicide is used continuously at lower than recommended rates. This allows a gradual increase in the population of weed biotypes with an increased ability to metabolize the compound.

The most likely way to cause evolution of resistant weed populations is by exerting selection pressure on weeds with the same herbicides over several generations. Using long soil residual herbicides, the same herbicide continuously, or rotating among herbicides that target the same site exert selection pressure for resistance over several generations.

Continuous planting of the same crop in each growing season reduces options for rotating to herbicides with a different target site. For example, crop rotation in California rice is difficult, so rice is planted continuously. The herbicide bensulfuron (Londax, an ALS-inhibiting herbicide) was registered in rice in California in 1989. It was highly effective on most rice weeds. There were few alternative control techniques used in rice so Londax was used

extensively for several years (Anonymous 1993). Resistance evolved quickly and now there are at least four weed species (Table 2) resistant to Londax.

The limited number of registered herbicides in many minor crops restricts the ability to rotate among compounds with different sites of action. This often leads to continuous use of one or a few herbicides and increases the probability of herbicide resistance evolving among the weed populations present in those fields. However, resistance has not become a problem in California's minor crop production areas. This is probably because of the extensive use of hand labor, cultivation and frequent rotation among a number of crops that have herbicides registered with different target sites. While hand labor and cultivation continue as effective methods to avoid resistance, herbicide rotation that has accompanied crop rotation may become ineffective since herbicides that target branched chain amino acid synthesis (sulfonylureas [ALS inhibitors] and imidazolinones [AHAS inhibitors]) are being registered for several of California's minor crops, including tomatoes and sugar beet. In addition, cotton, corn and alfalfa all have ALS-inhibiting and AHAS-inhibiting herbicides registered. The risk of weeds evolving resistance to these herbicides will increase if ALS-herbicides are used continuously in several crops within a rotation, since there will be continued selection pressure exerted on branch chain amino acid synthesis. Exclusive use of herbicides for weed control can rapidly select for resistance when other control practices such as tillage or hand hoeing are not used to control herbicide resistant weeds. In general, non-chemical methods will not select between susceptible and resistant plants and should be used whenever possible. Resistance also evolves more quickly in lower value solid-seeded crops grown on large acreage since cultivation and hand-weeding of these crops may not be feasible. Farmers with crops grown over large areas tend to rely heavily on herbicides for weed control. These large acreages contain a greater number of individual weeds that may contain a resistance trait.

## **Strategies for Prevention and Management**

Any management action that reduces the selection pressure for resistance will reduce the rate of resistance evolution. A number of papers have outlined various strategies that can be used to reduce the potential selection for herbicide resistant and to management herbicide resistant weed populations (Crites 1990, Shaner et al. 1992, Mallory-Smith et al. 1993, Retzinger and Mallory-Smith 1997). Crop rotation is one of the best tools to prevent resistance. Rotating to another crop allows the use of both chemical and non-chemical methods of control. Manipulation of planting time, crop competitiveness, cultivation techniques, hand weeding and herbicides with different target sites are all possible in a crop rotation system. Farmers and Pest Control Advisors (PCAs) in California use many of the methods listed above to control weeds. These characteristics of California agricultural production are probably the reason that few weed species have evolved herbicide resistance. As highly effective herbicides with the same target site become registered in California in multiple crops of a rotation, the risk of resistance evolution increases. Herbicides with different chemistries and trade names, but with the same target site, can reduce the effectiveness of herbicide rotation. Some common crop rotations include cotton, corn, tomato, sugarbeet and alfalfa. All these crops now have herbicides registered for use that target the same site (ALS). Weed species will evolve resistance rapidly without rotation of herbicides with different target sites.

The use of herbicide resistant crops is a new technology with increasingly widespread adoption. In many cases, growers will rely more heavily on a single herbicide in these cropping systems. Such a strategy will likely select for weed biotypes resistant to that herbicide or mode of action. Tank mixing, rotating herbicides, rotating from herbicide resistant to non-herbicide resistant crop varieties, as well as integrating non-chemical control options within the weed management program will reduce the potential for weed biotypes evolving a resistant trait.

The use of short-residual herbicides also reduces selection pressure for herbicide resistance. In addition, tank-mixing herbicides with different modes of action (Table 1) can inhibit evolution of resistance, but combinations should be used that broaden the spectrum of weeds controlled as well as control the weed species of major concern. If two herbicides have nearly the same weed control spectrum, it would be better to rotate between them rather than tank-mix the two compounds. It is unlikely, but theoretically possible, to select for resistance to two herbicides simultaneously.

While weeds have traits that enhance the possibility for evolution of resistance, they also have traits that reduce the chance of resistance evolving. Weed species with seeds that remain dormant in the soil for several years will maintain a population of susceptible plants within the seedbank. Maintaining susceptible plants in the population can dilute the resistance trait. If there is a fitness cost to resistance, such as in the case of triazine resistance, then removing the herbicide at some point in the crop rotation cycle allows for competition between the resistant and susceptible plants, further diluting the gene pool for the resistance trait.

Besides crop rotation, the use of certified seed, equipment sanitation, and cultivation and/or hand-weeding all impede resistance evolution. A resistance problem is usually not detected until land managers or farmers observe about 30% weed control failure for a particular species. If these resistant weed patches can be identified early before their populations increase, management practices can be employed to prevent their spread. If weed escapes appear in patterns, such as distinct strips, or if several species normally controlled by the herbicide are present in these skips, then the problem is probably associated with a calibration or application error. However, patches represented by only one escaped species showing no distinctive pattern may indicate a herbicide resistant population. Suspicious areas should be brought to the attention of a Farm Advisor or Extension Specialist, especially if weed populations reoccur in subsequent years following use of the same herbicide.

California weed management will change significantly with the introduction of new herbicides and the advent of herbicide resistant crops. If we maintain a selection pressure through continued use of these new tools, the new tools will soon be rendered ineffective. Adopting proactive management strategies to prevent herbicide resistance conserves important weed control tools. If resistance management strategies are ignored there is the potential that IPM systems may lose flexibility to deal with weed problems.

**Table 1. Herbicides, their mode of action, and herbicide classes registered for use in California.** Resistance has evolved in most groups and are listed as weeds worldwide, in the United States, and in California. Chemical families marked with the same shading pattern have been shown to result in cross-resistance among weed species.

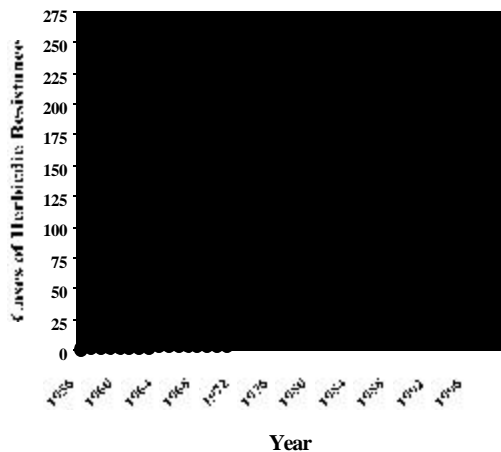
Mode of action	Trade name(s)	Common name	Chemical class	Number of resistant weed species		
				World-wide	United States	California
Photosynthesis inhibitor; electron diversion at photosystem I	Reward, Diquat Gramoxone, Cyclone, Starfire	Diquat Paraquat	Bipyridyliums	25	2	0
Photosynthesis inhibitors at photosystem II	Aatrex and others Bladex Pramitol Caparol and others Princep	Atrazine Cyanazine Prometon Prometryn  Simazine	Triazines	61	17	1
Photosynthesis inhibitors at photosystem II	Velpar, Pronone Sencor, Lexone	Hexazinone Metribuzin	Triazinones	3	1	1
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Stam, Stampede, Propanil	Propanil	Amide	2	1	0
Photosynthesis inhibitor at photosystem II; same site as triazines but different binding behavior	Basagran, Lescogran	Bentazon	Benzothiadiazole	0	0	0
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Betanex, Betamix Betanal, Betamix, Spin-Aid	Desmedipham Phenmedipham	Phenyl-carbamates	0	0	0
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Pyramin Tough	Pyrazon Pyridate	Pyridazinones	0	0	0
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Hyvar	Bromacil	Uracil	1	1	0
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Karmex, Direx and others Lorox Spike	Diuron  Linuron Tebuthiuron	Ureas	15	3	0
Photosynthesis inhibitors at photosystem II; same site as triazines but different binding behavior	Buctril, Moxy	Bromoxynil	Nitriles	1	1	0
Branched chain amino acid synthesis inhibitors at acetolactate synthase (ALS); also called acetohydroxyacid synthase (AHAS)	Arsenal, Stalker, Chopper Pursuit	Imazapyr  Imazethapyr	Imidazolinones	18	13	0
Branched chain amino acid synthesis inhibitors at acetolactate synthase (ALS); also called acetohydroxyacid synthase (AHAS)	Londax Telar, Glean Manage, Permit Accent Shade-Out, Matrix Oust Upbeet	Bensulfuron  Chlorsulfuron Halosulfuron Nicosulfuron Rimsulfuron Sulfometuron Triflusulfuron	Sulfonylureas	47	15	6

Branched chain amino acid synthesis inhibitors at acetolactate synthase (ALS); also called acetohydroxyacid synthase (AHAS)	Firstrate	Cloransulam	Triazolopyrimidine	2	2	0
Branched chain amino acid synthesis inhibitors at acetolactate synthase (ALS); also called acetohydroxyacid synthase (AHAS)	Staple	Pyriothiobac	Pyrimidinyloxybenzoates	1	0	0
Aromatic amino acid inhibitor at EPSP synthase	Roundup, Accord, Rodeo, Touchdown	Glyphosate	Glycine	2	1	1
Glutamine synthesis inhibitor	Finale, Liberty, Rely	Glufosinate	Phosphinic acid	0	0	0
Bleaching; inhibitor of carotenoid synthesis at phytoene desaturase (PDS)	Predict, Solicam, Zorial	Norflurazon	Pyridazinone	0	0	0
Bleaching; inhibitor of carotenoid synthesis at phytoene desaturase (PDS)	Sonar	Fluridone	Unclassified herbicide	0	0	0
Lipid synthesis inhibitors at acetyl CoA carboxylase (ACCCase)	Hoelon Whip, Acclaim Fusilade	Diclofop Fenoxaprop Fluazifop	Aryloxy phenoxy propionate	21	8	1
Lipid synthesis inhibitors at acetyl CoA carboxylase (ACCCase)	Prism, Select Poast, Vantage	Clethodim Sethoxydim	Cyclohexanedione	6	2	0
Lipid synthesis inhibitors; not ACCCase	Lasso, Partner Dual	Alachlor Metolachlor	Chloroacetamide	3	0	0
Lipid synthesis inhibitors; not ACCCase	Sutan Ro-Neet Eptam, Eradicane Ordram Tillam Bolero	Butylate Cycloate EPTC Molinate Pebulate Thiobencarb	Thiocarbamates	3	2	1
Lipid synthesis inhibition; not ACCCase	Prefar, Betasan	Bensulide	Acetamide	0	0	0
Fatty acid synthesis inhibitor	Nortron, Prograss	Ethofumesate	Benzofuran	1	1	0
Growth regulators; synthetic auxins (action similar to indoleacetic acid)	Banvel, Vanquish, Clarity	Dicamba	Benzoic acids	3	1	0
Growth regulators; synthetic auxins (action similar to indoleacetic acid)	Several Several Several	2,4-D MCPA Mecoprop (MCP)	Phenoxy carboxylic acids	15	3	0
Growth regulators; synthetic auxins (action similar to indoleacetic acid)	Transline, Stinger, Lontrel Garlon, Remedy, Pathfinder, Grandstand, Turflon	Clopyralid Triclopyr	Picolinic acids	1	1	0
Mitotic disruptors; microtubule assembly inhibitors	Balan Sonalan, Curbit Surflan Prowl, Pendulum Barricade, Endurance, Factor Treflan	Benefin Ethalfuralin Oryzalin Pendimethalin Prodiamine Trifluralin	Dinitroanilines	9	5	1
Mitotic disruptors; microtubule assembly inhibitors	Dimension Visor	Dithiopyr Thiazopyr	Pyridazine	1	0	0
Mitotic disruptor; microtubule assembly inhibitors; different site than dinitroanilines	Kerb	Pronamide	Benzamide	1	1	0

Cell division inhibitor; site unknown	Devrinol	Napropamide	Acetamide	0	0	0
Cell wall synthesis inhibitor	Gallery	Isoxaben	Benzamide	0	0	0
Cell wall (cellulose) synthesis inhibitor	Casoron, Norosac	Dichlobenil	Nitrile	0	0	0
Membrane disruptors	DMSA and others Several	DSMA MSMA	Organic arsenicals	1	1	0
Rapid membrane destruction; site unknown	Scythe	Pelargonic acid	Unclassified herbicide	0	0	0
Free radical generator; inhibitor of protoporphyrinogen oxidase (PPO)	Goal	Oxyfluorfen	Diphenyl ether	0	0	0
Free radical generator; inhibitor of protoporphyrinogen oxidase (PPO)	Ronstar	Oxadiazon	Oxadiazole	0	0	0
Free radical generator; inhibitor of protoporphyrinogen oxidase (PPO)	Milestone	Azafenidin	Triazolone	0	0	0
Unknown	Avenge	Difenzoquat	Pyrazolium salt	1	1	1
Unknown	Metam, Vapam	Metham	Dithiocarbamate	0	0	0
Unknown	Aquathol, Hydrothal, and others	Endothall	Disodium salt of methanearsonate	0	0	0
Unknown	Several Barespot Monobor-Chlorate	Copper sulfate and chelate Sodium chlorate and metaborate	Inorganics	0	0	0

**Table 2. Herbicide resistant weeds in California.**

Species	Common name	Area	Year reported	Chemical class (herbicide)
<i>Senecio vulgaris</i>	Common groundsel	Orchard, asparagus	1981	Triazine (atrazine)
<i>Lolium perenne</i>	Perennial ryegrass	Roadside, railway	1989	Sulfonylurea (sulfometuron)
<i>Cyperus difformis</i>	Smallflower umbrella sedge	Rice	1993	Sulfonylurea (bensulfuron)
<i>Sagittaria montevidensis</i>	California arrowhead	Rice	1993	Sulfonylurea (bensulfuron)
<i>Salsola tragus</i>	Russian thistle	Roadside	1994	Sulfonylurea (chlorsulfuron, sulfometuron)
<i>Avena fatua</i>	Wild oat	Barley, wheat	1996	Pyrazolium salt (difenzoquat)
<i>Ammania auriculata</i>	Redstem	Rice	1997	Sulfonylurea (bensulfuron)
<i>Scirpus mucronatus</i>	Ricefield bulrush	Rice	1997	Sulfonylurea (bensulfuron)
<i>Echinochloa phyllopogon</i>	Late watergrass	Rice	1998	Thiocarbamate (thiobencarb)
<i>Echinochloa phyllopogon</i>	Late watergrass	Rice	1998	Aryloxyphenoxy propionic acid (fenoxaprop)
<i>Lolium rigidum</i>	Rigid ryegrass	Orchard, roadsides	1998	Substituted amino acid (glyphosate)
<i>Echinochloa crus-galli</i>	Barnyardgrass	Cotton	1999	Dinitroaniline (trifluralin)



**Figure 1. Chronological increase in herbicide resistance in weeds worldwide.**

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