Herbicide Resistance Management Practices for Reed Canarygrass

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More than 115 reed canarygrass (RCG) germplasms (breeding lines) are registered in the United States and Canada, and the number of wild populations and cultivar-wild type hybrids has yet to be experimentally determined. A high level of genetic variability greatly increases the likelihood that some populations will evolve herbicide resistance. Resistance is the inherited ability to tolerate treatments or recover from treatment effects. Devine (1997) estimated that resistance to grass-specific herbicides (graminicides) could appear in some grass species after only 6 – 10 consecutive years of selection pressure. If RCG populations develop widespread resistance to graminicides, selective chemical control options will be lost as a management tool for RCG. Practicing resistance management will enable us to extend the functional life of graminicides for RCG suppression. The purposes of this article are to briefly outline the mechanisms of graminicide action and resistance, and to propose management practices that can be employed to delay the onset of resistance to graminicides. A starting point to develop a strategy for managing herbicide resistance is understanding the molecular mechanisms of herbicide toxicity and the genetics of resistance.

Molecular Mechanisms of Graminicide Action

Grass-specific herbicides of the aryloxy-phenoxypropionic acid (APP, common names end in "fop") and cyclohexane-1,3-dione (CHD, common names end in "dim") chemical families inhibit the metabolic enzyme acetyl coenzyme-A carboxylase (ACCase). ACCase catalyzes the formation of malonate, a building block of a variety of fatty acids, such as those that comprise the cell membrane. ACCase is composed of two different enzyme subunits joined into a single enzyme package. The production of malonate is a two-step reaction, and each enzyme subunit catalyzes one of the steps.

A normally functioning ACCase enzyme can assemble malonate because the three-dimensional shape of its active site (the part of the enzyme where the chemical reaction takes place) is similar to the three-dimensional shape of both components from which malonate is formed. APP and CHD herbicides bind to a portion of the enzyme other than the active site and change its shape so that it is unable to catalyze the reactions (Maier et al. 1994).

Graminicides are grass-specific because the AC-Case found in sedges, other monocots, and broadleaf species is *structurally different* than the AC-Case found in the grass family (Poaceae). Non-grasses are tolerant of APP and CHD herbicides because their ACCase lacks a binding site for the herbicide.

When graminicides interfere with grass ACCase activity, the cell runs out of the malonate building blocks with which it constructs lipids. Plant growth ceases almost immediately when the preexisting pool of malonate building blocks is depleted (this is why graminicides are observed to stunt RCG growth) and the actively growing portions of the plant and its rhizome system eventually succumb to tissue necrosis. Suppression of the inactive (dormant) portions of the plant requires additional herbicide applications.

The Genetic Basis of Herbicide Resistance

Repeated exposure to graminicides has produced resistant genotypes in at least 14 different grass species, including *Phalaris minor* (annual littleseed canarygrass). In 11 of these species (including *P. minor*), a direct link has been established between resistance and changes in ACCase and the DNA base pair sequences of the gene that encodes AC-Case. In each of these 11 species, different resistant genotypes displayed different patterns of resistance to different APP and CHD herbicides (Devine 1997, Gengenbach et al. 1999). In other words, if a genotype developed resistance to sethoxydim (CHD), it was not automatically cross-resistant to clethodim (CHD) or fluazifop (APP).

The three-dimensional shape of an enzyme influences how it interacts with other molecules, or if it will interact at all. The mutations that confer different resistance patterns occur in the region of the gene that codes for the three-dimensional structure of the sites to which the graminicides bind. Mutations in this coding region can result in a variety of amino acid substitutions, each altering the structure of the herbicide's binding site in a unique manner, and each leading to a specific resistance-susceptibility pattern. For example, the herbicide binding site in a susceptible plant can accommodate all APP and CHD herbicides and is susceptible to

all graminicides, but the mutant (tolerant) herbicide binding site may only be able to accommodate CHD molecules; APP molecules no longer fit. If the APP molecule no longer fits, it will no longer be able to change the three-dimensional shape of the ACCase enzyme and will not affect its ability to catalyze the reaction that yields malonate. The resistance pattern in this mutant would be CHD-susceptible, APP-resistant (Devine 1997).

At the local population level, repeated herbicide treatments will reduce or eliminate susceptible individuals, increasing over time the proportion of the population carrying resistance mutations in their DNA. Eventually, the population will consist entirely of resistant genotypes. Preventing or delaying this progression of events is a primary goal of herbicide resistance management.

Resistance-Susceptibility Patterns: Resistance Management Tool or Temporary Transition State?

It may seem as though the experimentally observed resistance-susceptibility patterns offer the opportunity to alternate APP and CHD herbicide formulations as a resistance management strategy. However, *these resistance-susceptibility patterns may only represent a temporary transition state between complete susceptibility and complete tolerance to all graminicides,* and may be an artifact of the short-term scope of inference of herbicide resistance investigations. Complete resistance to all graminicides may evolve rapidly from this state.

Predicting the rate of increase of resistance mutations in RCG populations is difficult because clonal growth may act to decrease the rate at which resistance evolves, while cultivated varieties of RCG may have higher rates of evolution because of enhanced seed production characteristics caused by selective breeding (Ostrem 1988, Sahramma et al. 2004). It remains unclear whether naturalized satellite populations of these cultivated genotypes will attain and pass on resistance alleles at a faster rate than "wild" field populations. We cannot be assured that randomly alternating APP and CHD herbicides will be an adequate resistance management practice in the long-term.

Resistance Management Practices for Reed Canarygrass

1. **Incorporate variability into your management plans**. Resistance will evolve more slowly with variable treatment approaches than with regular patterns and consistent treatment practices.

2. **Use an integrated, multiple-treatment approach to RCG control**. Do not rely exclusively on herbicide applications to restore a degraded plant community, but use other control methods in conjunction with herbicide applications. Apply herbicides at a lethal rate and according to label specifications. Hand pull scattered individuals. On sites with water control structures, couple prolonged flooding with other suppression treatments. Reintroduce stabilizing disturbances (such as controlled burns) to enhance growing conditions and provide germination requirements for replacement species. To accelerate control, couple tillage or prescribed flooding to herbicide applications. When control is accelerated, fewer herbicide applications are required and there is less time for selection of tolerant genotypes.

3. **Abate landscape disturbances prior to initiating herbicide applications**. RCG invasions are often a symptom of interacting chronic background disturbances (e.g., nutrient or stormwater inputs, sedimentation, hydrological modifications). Without correcting the underlying causes of invasion, probabilities of long-term, sustainable success are small and repeated herbicide applications will be required to sustain suppression.

4. **Reserve grass-specific herbicides** for high quality mixed-species stands with intact propagule banks of native species.

5. **Change chemical families of herbicides (in a random pattern).** If RCG occurs in a patchy or clumped distribution within a stand of native species, randomly substitute graminicide applications with directed applications of broad-spectrum herbicides (glyphosate or imazapyr). Where possible, use sequential applications of broad-spectrum herbicides (in early spring before native species emerge) and graminicides after native species emerge (late spring through late summer) when follow up treatments are necessary. If RCG is commingled with desirable native species and the risk of collateral damage is high, randomly alternate among the APP and CHD graminicides. Also, read and understand the herbicide label before applying graminicides; certain graminicide formulations should not be applied to some types of sites except during prolonged hydrological drawdown.

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6. **Practice active reintroductions of replacement species**. Treatment effects on RCG can be augmented by the presence of competing vegetation. Actively reintroduce replacement species. Refer to the reed canarygrass working group website (http://phalaris.pbwiki.com) for a list of species that may have potential to replace RCG.

7. **Apply herbicides under appropriate field conditions and with carefully selected additives** to ensure maximal effectiveness of the herbicide. Refer to *Plants out of Place 17:4-6 (2007)*.

8. **Regularly monitor results of herbicide applications and maintain detailed herbicide application records.** Look for trends, such as gradual declines in the degree of suppression achieved by herbicides. Determine if inclement weather, defective equipment, or improper application techniques could be responsible for poor herbicide performance. Consider an alternative herbicide formulation if extraneous factors influencing herbicide performance cannot be identified, and the desired level of suppression is no longer being achieved.

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