The Basics of Herbicide-Resistant Weeds

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My program at UC Davis is focused on management of weeds, especially herbicide-resistant weeds, in orchard and vineyard cropping systems. So, how does that apply to this California Weed Science Society session dedicated to managing weeds in roadsides, utilities, and industrial sites? Simple, although the details may differ slightly, the concepts of herbicide-resistance are pretty much the same regardless of whether herbicides are used in annual crops, perennial crops, or non-crop sites!

Herbicides can provide impressive levels of weed control in many crop and non-crop situations; however, not all weedy species are equally controlled due to varying levels of natural tolerance or evolution of herbicide-resistant weed biotypes. Herbicides impose a great degree of "selection pressure" on weed populations and if the same herbicide or herbicides with the same mode of action are used repeatedly, herbicide-resistant or -tolerant species can build up in the population after several generations

Herbicide tolerance and weed shifts: Weedy plants can be tolerant of herbicides due to a variety of temporal, spatial, or physiological mechanisms. For instance, a weed that emerges after a burn down herbicide is applied or completes its lifecycle before a post-emergence herbicide is applied may avoid control efforts. Similarly, large-seeded or perennial weeds can emerge from deeper in the soil and may avoid germinating in soil treated with a preemergence herbicide. Other weedy species have physiological mechanisms of tolerance and avoid control through reduced herbicide uptake or translocation, rapid detoxification, or insensitive target sites. Regardless of the mechanism of tolerance, repeated use of an herbicide can lead to "weed shifts" in which weed populations become dominated by species that are not affected by the weed control measures used.

Herbicide resistance: Herbicide resistance in weeds is an evolutionary process and is due in large part to selection with repeated use of the same herbicide or products with the same mode of action. Herbicides do not "cause" resistance; instead they select for naturally occurring resistance traits. On a population level, organisms occasionally have slight natural mutations in their genetics; some of these are lethal to the individual, some are beneficial, and some are neutral. Occasionally, one of these chance mutations affects the target site of an herbicide such that the herbicide does not affect the new biotype. Similarly, mutations can affect other plant processes in a way that reduces the plant's exposure to the herbicide due to reduced uptake or translocation or through more rapid detoxification. Whatever the cause, under continued selection pressure with the herbicide, resistant plants are not controlled and their progeny can build up in the population. Depending on the initial frequency of the resistance gene in the population, the reproductive ability of the weed, and competition, it may take several (or many) generations until the resistance problem becomes apparent.

Target-site resistance: Herbicides usually affect plants by disrupting the activity of a specific protein (enzyme) that plays a key role in plant biochemical process. Target site resistance occurs

when the target enzyme becomes less sensitive or insensitive to the herbicide. The loss of sensitivity is usually associated with a mutation in the gene coding for the protein and can lead to conformational changes in the protein's structure. These physical changes can impair the ability of one or more herbicides to attach to the specific binding site on the enzyme; thus reducing or eliminating herbicidal activity. Certain herbicide groups are particularly vulnerable to developing target site resistance, because resistance can be endowed by several mutations, thus increasing the probability of finding resistant mutants in weed populations - even in those not previously exposed to that herbicide group. For example, specific mutations resulting in seven different amino acid substitutions in the acetolactate synthase (ALS) gene are known to confer resistance to ALS-inhibiting herbicides in weed biotypes selected under field conditions. Something similar occurs with the grass herbicides that inhibit the enzyme acetyl coenzyme A carboxylase (ACCase) for which at least five point mutations causing amino acid substitutions within the gene are associated with cross-resistance patterns observed at the whole plant level involving four classes of ACCase inhibiting herbicides. The existence of so many mutations conferring resistance is reason resistance to these herbicides is frequently found and can evolve rapidly. Resistance to glyphosate can also be target-site mediated in some cases.

Non-target-site resistance: Several mechanisms confer resistance to herbicides without involving the active site of the herbicide in the plant. Of these, the best known is the case of metabolic resistance due to an enhanced ability to metabolically degrade the herbicide. Non-target-site resistance can evolve from the intensive use of diverse and unrelated selective herbicides that are similarly effective on a certain weed species and share a detoxification pathway or a mechanism precluding their accumulation at the target site (exclusion or sequestration) that is relatively common in plants. The management of non-target-site herbicide resistance often represents a greater challenge than target-site resistance because a simple change in herbicide mode of action may not alleviate the problem. Reduced herbicide absorption and/or translocation can contribute to resistance in certain biotypes. These have generally been accessory mechanisms that contribute towards resistance in addition to a major resistance mechanisms. However, recent evidence suggests that changes in absorption and/or translocation are an important contributor to glyphosate resistance in several weed biotypes.

Current status of herbicide-resistance in weeds: Herbicide resistant weeds are an issue around the world; but the greatest problems with resistance tend to be found in countries with highly industrialized agricultural cropping systems due to greater reliance on herbicides. Herbicide resistant weed biotypes have been reported in at least 60 countries and include about 396 unique species-herbicide group combinations worldwide. Herbicide resistant weeds around the world and throughout the U.S are dominated by the photosystem II inhibitors and by ALS inhibitors due to the widespread use of these diverse herbicide classes in broad acreage cereal and grain crops. Some of the most troubling herbicide resistant biotypes are multiple resistant biotypes – one population of rigid ryegrass in Australia is reported to be resistant to 9 different modes of action!

Management of herbicide-resistant weeds: A number of factors affect the degree of selection pressure for herbicide resistant weeds. However, if preventive measures are taken to reduce selection pressure, herbicide resistance can be avoided or delayed. As outlined previously, repeated use of the same herbicide or herbicides with the same mode of action can select for

weeds that are resistant or tolerant to that mode of action. As an herbicide controls the susceptible biotypes, with repeated use of the same herbicide, the resistant biotype gradually builds up in the population. Therefore, a major goal of herbicide resistance management is to reduce selection pressure. In this context, herbicide rotation and tank mixes become important resistance management tools and often are used as the first line of defense against the selection of herbicide-resistant weeds.

Non-crop areas such as roadsides, canal banks, and industrial sites have few crop rotational alternatives. Therefore, in these systems, rotation or tank mixes of herbicides with different modes of action should be a part of the management plan to prevent the buildup of weeds that are resistant to that particular mode of action. When herbicides with different modes of action are used in rotation or mixtures, the selection pressure for any one herbicide is reduced. Thus, the weeds will have difficulty adapting to this continuous alteration in selection pressure.

Studies have found that the selection pressure on susceptible weeds from herbicides with longer residual activities is higher than that from herbicides with shorter or no residual activities because one treatment can result in exposure of multiple weed cohorts (ie. flushes) to the herbicide. However, when herbicides with no residual activity are used multiple times in a season, selection pressure is equally high and can lead to selection for herbicide-resistant weeds as has been observed with glyphosate-only weed control programs. In fact, short-term residual herbicides in combination with post-emergence herbicides are being recommended for management of glyphosate-resistant weeds in many cropping systems.

Herbicide resistant weed conclusions: Resistance mitigation seeks to diversify weed control methods in order to delay the evolution process by reducing the selection pressure exerted through the use of herbicides. Target-site resistance is conferred by an alteration causing loss of plant sensitivity to herbicides with a specific mechanism of action. It is, therefore, clear that one way of dealing with the problem is by switching to another herbicide effective on the same weed species, but having a different mechanism of action. The use of herbicide mixtures or sequences involving herbicides with different mechanisms of action can protect the herbicides and delay the evolution of resistance to both, since mutants with resistance to one herbicide would be controlled by the other herbicide and vice-versa. However, the recurrent use of the same herbicide mixture could theoretically select for biotypes with resistance to both herbicides (multiple resistance).

Non-target-site resistance may involve different herbicides and the enhanced expression of mechanisms that are common in plants and thus easily selected for. If several herbicides share a common degradation route, such as the ubiquitous P450 monoxidation, their use will select for the same mechanism of resistance in biotypes that will be resistance to all even if these herbicides are used in mixtures or sequences with each other. Thus, combining or changing herbicides to control non-target-site-resistant biotypes becomes very difficult. Non-target-site resistance may involve the accumulation of genes contributing partial resistance levels.

From this discussion of resistance mechanisms in herbicide resistant weeds, it should be clear that resistance cannot be mitigated only by switching or combining herbicides in production systems that rely solely on the intensive use of selective herbicides for weed control. Instead, herbicide resistance management requires the integrated diversification of chemical and non-

chemical weed control methods to reduce selection pressure for resistant weed biotypes. Herbicides are one of the most effective tools for weed management; however, they must be used judiciously. They should be 'one of the many tools' in a weed management toolbox rather than the only tool, else we are at risk of losing effective herbicides due to the evolution of herbicide-resistant weeds.

For more resistance info: http://www.ipm.ucdavis.edu/IPMPROJECT/glyphosateresistance.html or the UCWeedScience blog at http://ucanr.edu/blogs/UCDWeedScience/index.cfm