Herbicide Resistance: Development and Management

Guide A-616

Jamshid Ashigh and Tracy M. Sterling[1]

Cooperative Extension Service • College of Agricultural, Consumer and Environmental Sciences

This publication is scheduled to be updated and reissued 1/15.

Worldwide, herbicides remain the most efficient technology for large-scale weed control. Therefore, the widespread evolution of herbicide resistance in weed populations within intensive crop production systems is a major threat to the sustainability and profitability of cropping systems. The introduction of new herbicides and herbicide modes of action to replace those herbicides failing due to resistance is essential for weed management. However, the rate of introduction of new herbicides for world agriculture has slowed dramatically. This is due to the difficulty and high cost involved in discovery and development of new herbicides and herbicide modes of actions, and, as a result, new herbicide or herbicide mode of action discovery and development will not occur at a rate required for proactive and reactive resistance management. Therefore, there is a strong imperative to use currently available herbicide resources in more sustainable ways (Walsh and Powles, 2004).

SCOPE OF THE PROBLEM

To date, resistance to 19 classes of herbicides has been reported in 330 biotypes of 189 species (113 dicots, 76 monocots) worldwide. In New Mexico, there are currently two confirmed cases of herbicide-resistant weed biotypes: a kochia (*Kochia scoparia*) biotype resistant to herbicides inhibiting acetohydroxyacid synthase (AHAS, also known as acetolactate synthase or ALS) and, more recently, a Palmer amaranth (*Amaranthus palmeri*) biotype resistant to glyphosate (Heap, 2009). Considering the widespread usage of glyphosate in the region (e.g., orchards, vineyards, industrial situations, and glyphosate-resistant crops [Roundup Ready crops]), the development of glyphosate resistance in prolific weeds such as Palmer amaranth is a threat to the future use of this important herbicide in agriculture. To date, sixteen prominent weed species have developed resistance to glyphosate worldwide. In the United States, nine weed species, including Palmer amaranth, common waterhemp (*Amaranthus rudis*), common ragweed (*Ambrosia artemisiifolia*), giant ragweed (*Ambrosia trifida*), hairy fleabane (*Conyza bonariensis*), horseweed (*Conyza canadensis*), Italian ryegrass (*Lolium multiflorum*), rigid ryegrass (*Lolium rigidum*), and johnsongrass (*Sorghum halepense*), have developed resistance to glyphosate, in several states (Heap, 2009).

RESISTANCE DEFINITIONS AND DEVELOPMENT

Herbicide resistance is the inherited ability of a plant to survive and reproduce following selection with a dose of herbicide normally lethal to the wild type of the plant. The development of herbicide resistance in weeds is an evolutionary process. Weed populations are extremely diverse genetically. In some cases, the genetic variation within weed populations includes the inherent abilities to resist some herbicides. However, the frequency of such variation in a normal weed population is very low.

However, if an herbicide is applied repeatedly on those populations (or herbicides from the same herbicide group are applied), the entire picture can change. As the majority of the susceptible biotypes are controlled after repeated applications, the few resistant biotypes are provided with a unique opportunity to proliferate. Therefore, the use of an herbicide (or herbicides from the same herbicide group) continuously for many years can drastically decrease the number of susceptible biotypes within the natural weed population and dramatically increase the number of resistant biotypes. In response to widespread use of a particular family of herbicides, weed populations can change in genetic composition such that the frequency of resistance gene(s) and resistant individuals increases. Thus, weed populations become adapted to the intense selection imposed by herbicides (Jasieniuk et al., 1996).

[1]Respectively, Extension Weed Specialist/Assistant Professor, Department of Extension Plant Sciences; and Professor, Department of Entomology, Plant Pathology, and Weed Science, both of New Mexico State University.

MECHANISMS OF HERBICIDE RESISTANCE IN WEEDS

Mechanisms of herbicide resistance include altered site of action, overproduction of the site of action, enhanced herbicide metabolism, decreased herbicide absorption and translocation, and herbicide sequestration. The most common mechanisms that cause herbicide resistance in weeds are altered site of action and enhanced herbicide metabolism. Site of action is the specific process within a plant that a particular herbicide or herbicide group inhibits. Alterations in the site of action that prevent the herbicide from binding are the most common mechanism of resistance (Heap and LeBaron, 2001). Enhanced herbicide metabolism is the superior ability of plants to convert the herbicide molecule to a form that is no longer toxic, whereas susceptible species are unable to detoxify the herbicide at the same rate as resistant species (Saari et al., 1994).

TYPES OF RESISTANCE

In some cases, resistant weeds can also survive the application of herbicides other than the herbicide to which they have developed resistance (i.e., the selecting herbicide). In such cases, resistant weeds are considered to have cross- or multiple resistance. Cross-resistance occurs when one resistance mechanism (e.g., enhanced herbicide metabolism) allows the plant to withstand herbicides from different chemical classes. However, when a plant has multiple resistance it possesses two or more distinct resistance mechanisms (e.g., two or more altered sites of action), which allow the plant to resist herbicides from different chemical classes (Hall et al., 1994). For example, a population of smooth pigweed (*Amaranthus hybridus*) from Illinois has resistance to atrazine, a photosynthesis-inhibiting herbicide; multiple resistance to primisulfuron (Beacon), a sulfonylurea herbicide; and cross-resistance to imazamox (Raptor), an imidazolinone herbicide (Maertens et al., 2004).

On the contrary, in some weeds, resistance to one herbicide results in increased susceptibility to another herbicide or other abiotic factors, such as standard cultivation practices, and/or biotic factors, such as effects of insect pests or infection by viruses and fungi. This phenomenon is known as negative cross-resistance (Gressel and Segel, 1990) and could be exploited in some resistant weed species. For example, Salhoff and Marton (1986) reported that triazine resistant *Kochia* biotypes from Idaho were more sensitive to 2,4-D than susceptible biotypes.

FACTORS AFFECTING RATE OF RESISTANCE DEVELOPMENT

Selection Pressure. Selection pressure is one of the most important determinants of resistance development. Major factors contributing to selection pressure include the efficacy of the herbicide and the frequency of use (Maxwell and Mortimer, 1994). In general, selection pressure is a measure of the ability of an herbicide to differentiate between susceptible and resistant plants. Therefore, herbicides with higher efficacy in the control of a specific weed population impose higher selection pressure for selecting the resistant individuals among that weed population. Moreover, depending on the soil and herbicide chemical properties, herbicide residual activity could also be a major factor in the development of herbicide resistance (Jasieniuk et al., 1996; Beyer et al., 1988). This means that the selection pressure on susceptible weeds from herbicides with longer residual activities would be higher than that from herbicides with shorter or no residual activities.

Initial Frequency of Resistance Mutations. Precise studies have not been performed on the initial frequency of resistance mutations in weeds. However, some estimates have shown that approximately one out of a billion seeds from a population could be resistant (Harms and DiMaio, 1991). While these mutations are rare, large weed populations increase the likelihood that they might be present.

Gene Flow. Pollen and seed movement are the major methods of gene flow between and within plant populations (Maxwell and Mortimer, 1994). Furthermore, the spread of resistance occurs more rapidly in cross-pollinated species compared to self-pollinated species (Jasieniuk et al., 1996). Therefore, resistance genes most likely arise in an area through mutation, and gene flow would facilitate the spread of the resistance genes among individuals within that area (Jasieniuk et al., 1996; Ashigh et al., 2008).

Fitness. Fitness is a measure of survival and ability of a given genotype (e.g., herbicide-resistant biotypes) to produce viable offspring in competition with the wild type (e.g., herbicide-susceptible biotypes) (Gressel, 2002). Although herbicide-resistant biotypes should be less fit than susceptible biotypes, depending on the mechanism of resistance and the environmental conditions, the fitness of resistant populations may vary. For example, under agricultural field conditions, triazine-resistant biotypes have been shown to be less fit than the susceptible biotypes, but many studies have not been able to detect fitness penalty in biotypes resistant to AHAS inhibitors (e.g., Pursuit) under those conditions (Ashigh and Tardif, 2009). The existence of fitness penalty under field conditions could be exploited for management of those resistant biotypes affected by it.

CONFIRMATION OF RESISTANCE

Resistant biotypes can be confused with weed escapes that result from herbicide failures caused by bad weather, type and size of the weeds, and/or improper applications. However, herbicide resistance should not be suspected unless an herbicide failure fits the following traits: (a) the same herbicide was used year after year; (b) one weed, which normally should be controlled, is not controlled, although other susceptible weeds are controlled; (c) a patch of one weed species that survived herbicide application is spreading; and (d) healthy weeds are mixed with controlled weeds of that same species. In cases where a control failure exhibits the above-mentioned traits, seeds of suspected herbicide-resistant weeds can be sent to the Plant Diagnostic Clinic at NMSU to the addresses below for confirmation of resistance.

Mailing address
Plant Diagnostic Clinic
MSC 3AE
New Mexico State University
P.O. Box 30003
Las Cruces, NM 88003-8003

Shipping address (UPS or FedEx)
New Mexico State University
Attn: Plant Diagnostic Clinic
945 College Ave.
Skeen Hall, Room N140
Las Cruces, NM 88003-8003

PREVENTION AND MANAGEMENT OF RESISTANCE

As resistance is generally the consequence of using a single herbicide repeatedly, any proactive or reactive approach should take an opposite view: use a diversity of methods to avoid repetition as much as possible.

Identification. Most populations of resistant weeds do not become apparent to growers until at least 10 to 30% of the weed population becomes resistant. Resistant populations can be identified with diligent field monitoring. Early detection enables growers to contain and manage the resistance problem sooner and to employ preventative operational measures to control the spread of resistance.

Prevention. Once a resistance problem is identified, the problem could be contained with restricted movement of crops and equipment. There have been cases where resistance was found in a field where previous practices should not have lead to resistance. In such cases, introduction through harvesting equipment (custom combining) could be the cause. This generally happens more in areas where resistance has already developed extensively. Therefore, biosanitary practices, such as cleaning equipment and removing and destroying resistant plants to prevent re-infestation of the field with resistant seed or plant parts, can be employed.

Cultural Control. The weeds associated with different crops differ due to differences in the competitiveness and life cycles of crops. Crop rotations and/or use of competitive cover crops work to suppress certain weeds. Rotation of crops may also permit the use of different chemical (i.e., use of herbicides from different modes of action), fertilization, and tillage programs.

Mechanical Control. Cultivation and hoeing provide weed control, which reduces reliance on herbicides. However, depending on the soil type, cultivation could increase the risk of soil erosion.

Chemical Control. The use of herbicide rotations and mixtures is frequently advocated for resistance prevention and management. However, herbicide rotations and mixtures should include compounds from classes of herbicides with different modes of action that control similar spectra of weeds. They should possess the same persistence in the environment and degrade in different manners. Recent studies have shown that herbicide mixtures are superior to herbicide rotations in resistance prevention and management among weed populations. The idea behind the use of herbicide mixtures and/or rotations is to reduce the selection of resistant individuals in a weed population with a single herbicide or herbicide family.

ACKNOWLEDGMENTS

Critical reviews of this article by Dr. François Tardif and Mr. Greg T. Bettmann are acknowledged.

REFERENCES

Ashigh, J., and F.J. Tardif. 2009. An amino acid substitution at position 205 of acetohydroxyacid synthase reduces fitness under optimal light in resistant populations of *Solanum ptychanthum. Weed Research*, *49*, 479–489.

Ashigh, J., I. Rajcan, and F.J. Tardif. 2008. Genetics of resistance to acetohydroxyacid synthase inhibitors in populations of eastern black nightshade (*Solanum ptychanthum*) from Ontario. *Weed Science*, *56*, 210–215.

Beyer, E.M., M.J. Duffy, J.V. Hay, and D.D. Schleuter. 1988. Sulfonylurea herbicides. In P.C. Kearney and D.D. Kaufmann (Eds.), *Herbicides: Chemistry, degradation, and modes of action* (pp. 117–189). New York: Marcel Dekker.

Diggle, A.J., P.B. Neve, and F.P. Smith. 2003. Herbicides used in combination can reduce the probability of herbicide resistance in finite weed populations. *Weed Research*, *43*, 371–382.

Gressel, J. 2002. *Molecular biology of weed control.* New York: Taylor & Francis, Inc.

Gressel, J., and L.A. Segel. 1990. Modeling the effectiveness of herbicide rotations and mixtures as strategies to delay or preclude resistance. *Weed Technology*, *4*, 186–198.

Hall, L.M., J.A.M. Holtum, and S.B. Powles. 1994. Mechanisms responsible for cross resistance and multiple resistance. In S.B. Powles and J.A.M. Holtum (Eds.), *Herbicide resistance in plants, biology and biochemistry* (pp. 243–263). Boca Raton, FL: Lewis Publishers.

Harms, C.T., and J.J. DiMaio. 1991. Primisulfuron herbicide-resistant tobacco cell lines: Application of fluctuation test design to *in vitro* mutant selection with plant cells. *Journal of Plant Physiology*, *137*, 513–519.

Heap, I. 2009. International survey of herbicide resistant weeds [Online]. Retrieved December 15, 2009 from www.weedscience.org

Heap, I., and H.M. LeBaron. 2001. Introduction and overview of resistance. In S.B. Powles and D.L. Shaner (Eds.), *Herbicide resistance and world grains* (pp. 1–22). Boca Raton, FL: CRC Press.

Jasieniuk, M., A.L. Brulé-Babel, and I.N. Morrison. 1996. The evolution and genetics of herbicide resistance in weeds. *Weed Science*, *44*, 176–193.

Maertens, K.D., C.L. Sprague, P.J. Tranel, and R.A. Hines. 2004. A*maranthus hybridus* populations resistant to triazine and acetolactate synthase-inhibiting herbicides. *Weed Research*, *44*, 21–26.

Maxwell, B.D., and A.M. Mortimer. 1994. Selection of herbicide resistance. In S.B. Powles and J.A.M. Holtum (Eds.), *Herbicide resistance in plants, biology and biochemistry* (pp. 1–27). Boca Raton, FL: Lewis Publishers.

Saari, L.L., J.C. Cotterman, and D.C. Thill. 1994. Resistance to acetolactate synthase inhibiting herbicides. In S.B. Powles and J.A.M. Holtum (Eds.), *Herbicide resistance in plants, biology and biochemistry* (pp. 141–170). Boca Raton, FL: Lewis Publishers.

Salhoff, C.R., and A.R. Marton. 1986. *Kochia scoparia* growth response to triazine herbicides. *Weed Science*, *34*, 40–42.

Walsh, M., and S.B. Powles. 2004. Herbicide resistance: An imperative for smarter crop weed management. In *Proceedings of the 4th International Crop Science Congress, Brisbane, Australia*.

January 2010 **Las Cruces, NM**