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Herbicide resistance in kochia: From single to multiple resistance

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ABSTRACT

Herbicide resistance in weeds is evolving rapidly worldwide complicating weed management and threating agricultural sustainability and food security. Resistance has been reported to all known herbicide modes of action and no new mode of action has been marketed in the past 25 years. Though most reported cases of resistance involve a single herbicide site of action, multiple-site resistance is increasing. As an example of the progression from single to multiple site resistance, this paper reviews the evolution and implications of herbicide resistance in kochia [*Kochia scoparia* (L.) Schrad.), a common and economically important weed in the North American Great Plains.

Key words: ALS inhibitors, Glyphosate, Kochia, Herbicide resistance, Photosystem II inhibitors, Synthetic auxins

Early literature documents agriculturalists using various naturally occurring substances and by-products to control various plant pests, including weeds, and Romans applied salt to fields of enemies to prevent growing of crops (Timmons 1970, Smith and Secoy 1976). However, chemical weed control in agricultural crops and tree fruits is considered to be of recent origin, dating from the mid-nineteenth century when lime and salt were recommended for weed control in Europe. The modern era of chemical weed control began with the discovery and development of chlorophenoxy acetic acid herbicides in the early 1940s (Peterson 1967, Timmons 1970). This family of herbicides, 2,4-D and MCPA in particular, not only transformed agriculture by revolutionizing weed control, but gave rise to the discipline of weed science and an entire industry.

The chlorophenoxy chemical family and several additional classes of herbicides developed since 1940 have provided highly effectively weed control selectively in many crops, resulting in more efficient production and higher crop yields compared with hand weeding and/or cultivation. Currently, the vast majority of cropland hectares in developed countries, and increasingly those in under-developed countries, are treated with chemical herbicides annually. Because all natural weed populations may contain very low frequencies of individual plants (biotypes) that are naturally resistant to certain herbicides, an unintended consequence of extensive herbicide use few people initially anticipated was that frequent repeated use of any herbicide could lead to shifts in species composition of weed populations and select for tolerant or resistant biotypes.

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Harper (1957) was among the first to warn of this evolutionary possibility. Numerous studies have since confirmed Harper's (1957) early prediction (Haas and Streibig 1992, Westra *et al*. 2004, Culpepper 2006, Wilson *et al*. 2007). Repeatedly using any single herbicide mechanism of action without alternative management tactics will eventually eliminate susceptible species or biotypes from an existing population and allow naturally tolerant or resistant biotypes to flourish and dominate the population (Gressel and Segel 1978, Maxwell and Mortimer 1994). Herbicide resistance is now widely recognized as the result of adaptive evolution of weed populations to intense selection pressure imposed by herbicides. Several recent reviews have documented the evolution of herbicide resistance (Powles and Yu 2010, Mithila *et al*. 2011, Burgos *et al*. 2013, Delye *et al*. 2013, Shaner 2014).

Genetic diversity is the heritable genetic variation within and among populations of species. Species with high genetic diversity, especially those that produce large quantities of seed that readily germinate, adapt and evolve faster in response to changing environmental conditions and selection pressures than species with low genetic diversity. Thus, species with high genetic diversity are prone to evolved resistance to herbicides. There is more than one mechanism of resistance for most herbicide modes of action and several known amino acid substitutions within target site proteins that prevent herbicide binding and disruption of critical biochemical pathways. Non-target site resistance mechanisms (*e.g*. reduced herbicide uptake or translocation, herbicide sequestration, or enhanced

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metabolism) allow plants to survive by preventing herbicide from reaching the target site or by producing more of the targeted enzyme than the herbicide can inhibit (*e.g.* over expression or gene amplification).

Currently, herbicide resistance has been confirmed in 247 weed species in 66 countries with evolved resistance to 22 of the 25 known herbicide sites of action (Heap 2015). Developed countries in which in most arable hectares are treated with herbicides have the greatest number of weed species resistant to known herbicide sites of action. Most reported cases involve resistance to a single herbicide site of action; however, several major weeds have evolved resistance to two or more sites of action. One example is Kochia, a broad-leaved weed of great economic importance throughout the North American Great Plains. This paper reviews the evolution and implications of herbicide resistance in kochia to raise awareness of the increasing threat of multiple herbicide resistance in weeds.

Herbicide resistance in kochia

kochia is a drought-tolerant annual forb with C_4 photosynthetic pathway believed to have been introduced into the United States from Eurasia as an ornamental in the late 1800s (reviewed in Friesen *et al.* 2009). This alien species is highly adaptive and drought-tolerant. Herbarium collections reveal kochia's rapid westward expansion in the North Western United States. Forcella (1985) speculated that kochia's exponential spread into Montana and Wyoming from 1940 to 1960 may have been facilitated by the chemical displacement of 2,4-D-sensitive species from their weed niches and replaced with 2,4-D tolerant taxa, such as kochia. Today, kochia is a major weed in agronomic crops and disturbed non-cropland areas throughout semi-arid and arid regions of the western United States and South-western Canada (Friesen *et al.* 2009).

High genetic diversity and short seed longevity in soil increase the probability of rare herbicide resistant biotypes within a population. Genetic diversity in kochia was found to be very high with greater proportion of diversity within populations than between populations (Mengistu and Messersmith 2002). High genetic diversity in kochia is maintained through substantial gene flow within and between populations by way of protogynous flowering, facultative open pollination, and tumbleweed mode of seed dispersal over long distances (Eberlein and Fore 1984, Stallings *et al.* 1995). Kochia is prone to evolved herbicide resistance, currently having evolved resistance to four modes of action and several cases of multiple site of action resistance.

Photosystem II inhibitors (groups C1 and C2)

Several active ingredients in the triazine family of herbicides (Group C1) were developed in the mid- and late-1950s and have been used in numerous agronomic crops, pastures, tree fruits and forestry, and on industrial sites. Some of those herbicides, such as atrazine and simazine, are still widely used today. As early as the mid-1970s, kochia growing along rail road embankments in Idaho and other Central and Western states was no longer controlled by triazine herbicides after many years of use for complete vegetation control (Bandeen *et al.* 1982). Those populations were found to be resistant to all commercial symmetrical-triazine herbicides and they rapidly spread to adjacent cropland, especially maize fields which usually were treated with atrazine or simazine. Within the following decade triazine-resistant kochia was present in several Midwest U.S. states along with several other major broad-leaved species in numerous states and other countries (Heap 2015). Additionally, kochia along rail road rights-of-way in North Dakota and Minnesota with resistance to diuron and tebuthiuron (Group C2) and metribuzin (Group 1) was discovered in 2004 (Mengistu *et al.* 2005). Resistance to triazine and triazinone herbicides most often is due to a target-site mutation (*e.g.* $serine_{264}$ to glycine substitution) which interferes with herbicide binding on the D1 protein in photosystem II, thereby inhibiting photosynthesis. However, resistance to diuron, tebuthiuron and metribuzin in kochia was due to a valine to isoleucine substitution at residue 219 of the psbA target-site in some plants and the more common $serine_{264}$ to glycine substitution in other plants (Mengistu *et al.* 2005).

ALS inhibitors (group B)

Sulfonylureas are a family of herbicides first commercialized for use in wheat and barley crops in 1982, and later in many crops. Also, in this group is the imidazolinone family consisting of six herbicides used in cereal and legume crops, forestry, non-cropland and on imidazolinone-resistant maize, rice, canola, sunflower, and wheat. Herbicides in this group kill weeds by inhibiting the enzyme acetolactate synthase (ALS) necessary for biosynthesis of amino acids essential for plant growth. Cereal grain and many other crops are able to metabolize sulfonylureas, whereas susceptible weeds and non-imidazolinone-resistant crops cannot. In 1987, selection of kochia and prickly lettuce (*Lactuca*

serriola L.) biotypes resistant to sulfonylurea herbicides in Kansas (Primiani *et al.* 1990) and Idaho (Mallory-Smith *et al.* 1990) wheat fields, respectively, was confirmed after as few as five consecutive years of sulfonylurea herbicide use. Evolved resistance to ALS-inhibiting herbicides in multiple species increased at an alarming rate, including ALS-resistant kochia in 13 U.S. states and Canadian provinces within seven years and nearly 150 species worldwide within 25 years after commercialization (Heap 2015). Target-site-based resistance resulting from several known amino acid substitutions in the conserved region of the ALS enzyme is the most common resistance mechanism in ALS inhibitor-resistant weeds.

Synthetic auxins (group O)

As a result of the reduced efficacy of ALS-inhibiting herbicides, producers began using dicamba extensively to control ALS-resistant kochia. It was not surprising then when in 1994 numerous kochia plants were not controlled with field use rates of dicamba in a maize field in Nebraska and in wheat fields in Northern Montana following several years of extensive dicamba use in cereal grain crops (Cranston *et al.* 2001). Testing of progeny of uncontrolled kochia plants in Montana revealed the frequency of plants producing resistant progeny was very low, but resistant biotypes were four- to five-fold more resistant than susceptible biotypes and the resistance could not be attributed to differential herbicide absorption, translocation, or metabolism. These findings led the authors' to speculate that dicamba resistance is a qualitative trait. More than 15-years later, Crespo *et al.* (2014) reported injury among 67 Nebraska kochia accessions treated with a 560 g/ha dose of dicamba ranged from 23 to 78% at 21 days after treatment. Furthermore, there was an 18-fold difference in dicamba dose required to achieve 90% injury between the least and most susceptible of four accessions selected from the larger group. In a similar study of 34 kochia accessions mostly from Kansas collected in 2012, there was an eight-fold difference in plant dry weight reduction five weeks after plants were treated with 420 g/ha of dicamba (Brachtenbach 2015). Results from the Nebraska and Kansas studies substantiate producer reports of reduced dicamba effectiveness for kochia control. The physiological, biochemical, and molecular basis for dicamba resistance in kochia has been studied extensively, but the precise mechanism(s) have not yet been determined (Mithila *et al.* 2011).

EPSPS synthase inhibitor

Glyphosate has been used extensively for many years for preplant burndown and to control volunteer crop plants and most grass and broad-leaved weeds in Great Plains cropping systems. The area treated with glyphosate and the total amount applied annually increased dramatically following the rapid adoption of glyphosate-resistant (GR, Roundup Ready®) crops. Glyphosate's broad spectrum effectiveness and relatively inexpensive cost attracted producers to often use glyphosate exclusively, especially in GR crops. These use patterns contributed to intense selection pressure on weed species, including Kochia, to evolve resistance to glyphosate.

Multiple failures to control kochia with glyphosate were first reported in Kansas in 2007 and subsequently were confirmed to be the first cases of glyphosate resistance in kochia (Waite *et al.* 2013, Heap 2015). More than 10 widely dispersed kochia populations in Kansas were confirmed resistant to glyphosate in 2010, with several other populations likely resistant but unconfirmed (Godar *et al.* 2015b). Resistance levels ranged from 3- to 11-fold based on greater EPSPS gene copy number compared to a susceptible population. Resistance to glyphosate due to gene amplification was first reported by Gaines *et al.* (2010).

By the end of 2012, GR kochia was widespread throughout the central Great Plains and also was confirmed that year in northern portions of the Great Plains extending into Canada. Currently, presence of GR kochia populations has been confirmed in 10 Great Plains states (Colorado, Kansas, Idaho, Montana, Nebraska, North Dakota, Oklahoma, Oregon, South Dakota, and Texas) and three Canadian provinces (Alberta, Saskatchewan and Manitoba) (Heap 2015). The distribution of GR kochia continues to expand into additional states and provinces.

Multiple site-of-action resistance

Because each of the herbicide groups mentioned above are commonly used in Great Plains cropping systems, it was only a matter of time until multiple site resistance evolved in kochia. The first reported cases of multiple resistance were along railroads in Illinois and in crop fields in Indiana where kochia was resistant to ALS- and photosystem II-inhibitors (Heap 2015). However, kochia is not a major agronomic weed in Midwest states, so the economic impact likely was not great. Conversely, the discovery of

ALS inhibitor- and glyphosate-resistant kochia in western Canada in 2012 (Beckie *et al.* 2013, Hall *et al.* 2013) arguably has had much greater economic impact and poses a threat to sustainable farming systems in the Northern Great Plains where kochia is a major agronomic weed and where ALS resistance is widespread (Beckie *et al.* 2013). The following year (2013), weed scientists in Kansas confirmed the first known case of resistance to four groups of herbicides (atrazine, group C1; chlorsulfuron, group B, glyphosate, group G; and dicamba, group O) in a single kochia population (Varanasi *et al.* 2015). The ratio of R:S plants to individual herbicides varied from 25% of plants resistant to atrazine to more than 85% of plants resistant to field use rates of chlorosulfuron, dicamba, and glyphosate. Resistance to atrazine and chlorsulfuron was due to target-site mutations in psbA and ALS genes, respectively, and resistance to glyphosate was due to EPSPS gene amplification. The mechanism of resistance to dicamba has not been determined.

Implications of evolved herbicide resistance

Economic considerations are a major criterion for most producers in making weed management decisions. Many producers are reluctant to proactively change effective weed management practices to more complex and/or expensive practices as long as current practices are still effective. Often the first reactive response to ineffective weed control is to increase herbicide use rate. In response to declining glyphosate effectiveness on kochia, Kansas producers increased glyphosate use rates from an average of 0.8 to 1.2 kg/ha and increased application frequencies from 2.0 to 2.9 during the years before discovery of GR-kochia in 2007 to 2012 (Godar *et al.* 2015a). During that same time period, Kansas producers reduced the exclusive use of glyphosate on GR crops from 49 to 15% of fields and began diversifying weed management practices. Clearly, the spread of GR kochia forced changes in practices and increased costs of weed management.

Canadian researchers have concluded the presence of GR weeds will increase environmental impact of weed management by requiring additional herbicides or by growers resorting to tillage to control GR weeds, the latter resulting in reduced soil quality and increased fossil fuel consumption (Beckie *et al.* 2014). The predicted environmental impact of increased tillage is supported by results of a visual survey of 1500 winter wheat stubble fields in Western Kansas in late August 2011 (Stahlman *et al.* 2011). Survey found 64% of wheat stubble fields had been sprayed with herbicide(s) to control weeds post-harvest and 31% of the fields had been tilled. Some of the tilled fields had been tilled after earlier herbicide treatment failed to control kochia. Poor herbicidal control of kochia in many fields and higher-than-expected percentage of tilled fields indicate a shift to more tillage to control herbicide-resistant kochia following wheat harvest. Evolution of weed resistance to herbicides not only complicates weed management but also threatens sustainable agricultural production and soil and water conservation gains achieved during past decades (CAST 2012).

REFERENCES

Bandeen JD, Stephenon GR and Cowett ER. 1982. Discovery and distribution of herbicide-resistant weeds in North America, pp. 9-30. In: *Herbicide Resistant Plants.* (Eds. Le Baron HM and Gressel J). John Wiley & Sons, New York.

Beckie HJ, Blackshaw RE, LR, Hall LM, Saunder CA, Martin S, Brandt RN and Shirrff SW. 2013. Glyphosate- and acetolactate synthase inhibitor-resistant kochia (*Kochia scoparia*) in Western Canada. *Weed Technology* **61**: 310-318.

Beckie HJ, Sikkema PH, Soltani N, Blackshaw RE and Johnson EN. 2014. Environmental impact of glyphosate-resistant weeds in Canada. *Weed Technology* **62**: 385-392.

Brachtenbach DA. 2015. kochia *scoparia response to dicamba and effective management practices for soybeans*. M.S. Thesis. Kansas State University, Manhattan. 53 p.

Burgos NR, Tranel PJ, Strieberg JC, Davis VM, Shaner D, Norsworthy JK and Ritz-Burgos C. 2013. Review: Confirmation of resistance to herbicides and evolution of resistance levels. *Weed Science* **61**: 4-20.

CAST (Council for Agricultural Science and Technology). 2012. *Herbicide-resistant Weeds Threaten Soil Conservation Gains: Finding a Balance for Soil and Farm Sustainability.* Issue Paper 49. CAST, Ames, Iowa.

Cranston HJ, Kern AJ, Hackett JL, Miller EK, Maxwell BD and Dyer WE. 2001. Dicamba resistance in *Kochia*. *Weed Science* **49**: 164-170.

Crespo RJ, Berhards ML, Sbatella GM, Kruger GR, Lee DJ and Wilson RG. 2014. Response of Nebraska kochia (*Kochia scoparia*) accessions to dicamba. *Weed Technology* **28**: 151-162.

Culpepper AS. 2006. Glyphosate-induced weed shifts. *Weed Technology* **20**: 277-281.

Delye C, Jasieniuk M and Le Corre V. 2013. Deciphering the evolution of herbicide resistance in weeds. *Trends in Genetics* **29**: 649-658.

Eberlein C and Fore Z. 1984. Kochia biology. *Weeds Today* **15**: 5-6.

Forcella F. 1985. Spread of kochia in the North Western United States. *Weeds Today* **16**: 4-6.

Friesen LF, Beckie HJ, Warwick SI and Van Acker RC. 2009. The biology of Canadian weeds. 138. Kochia *scoparia* (L.) Schrad. *Canadian Journal of Plant Science* **89**: 141-167.

Gaines TA, Zhang W, Wang D, Bukun B, Chisholm ST, Shaner DL, Nissen SJ, Patzoldt WL, Tranel PJ, Culpepper AS, Grey TL, Webster TM, Vencill WK, Sammons RD, Jiang J, Preston C, Leach JE and Westra P. 2010. Gene amplification confers glyphosate resistance in *Amaranthus palmeri*. *Proceedings National Academy of Science USA* **107**: 1029–1034.

Godar AS and Stahlman PW. 2015a. Consultant's perspective on the evolution and management of glyphosate-resistant kochia (*Kochia scoparia*) in Western Kansas. *Weed Technology* **29**: 318-328.

Godar AS, Stahlman PW, Jugulam M and Dille JA. 2015b. Glyphosate-resistant kochia in Kansas: EPSPS gene copy number in relation to resistance levels. *Weed Science* **63**: 587-59.

Gressel J and Segel LA. 1978. The paucity of plants evolving genetic resistance to herbicides: Possible reasons and implications. *Journal of Theoretical Biology* **75**: 349-371.

Haas H and Streibig JC. 1982. Changing patterns of weed distribution as a result of herbicide use and other agronomic factors. p 57-79. In: *Herbicide Resistant Plants*. (Eds. LeBaron HM and Gressel J). John Wiley & Sons, New York.

Hall, LM, Beckie HJ, Low R, Shiriff SW, Blackshaw RE, Kimmel N and Neeser C. 2013. Survey of glyphosate-resistant kochia (*Kochia scoparia* L. Schrad.) in Alberta. *Canadian Journal of Plant Science* **94**: 127-130.

Harper, JL. 1957. Ecological aspects of weed control. *Outlook on Agriculture* **1**: 197-205.

Heap I. The International Survey of Herbicide Resistant Weeds. Online. Internet. Wednesday, Accessed on November 28, 2015. *Available at www.weedscience.org*.

Mallory-Smith CA, Thill DC, and Dial MJ. 1990. Identification of sulfonylurea herbicide-resistant prickly lettuce (*Lactuca serriola*). *Weed Technology* **4**: 163-168.

Maxwell BD and Mortimer AM. 1994. Selectino for herbicide resistance, pp. 1-25. In: *Herbicide Resistance in Plants: Biology and Biochemistry*. (Eds. Powles SB and Holtum JAM). Lewis, Boca Taron, FL.

Mengistu LW, Christoffers MJ and Lym RG. 2005. A psbA mutation in kochia *scoparia* (L.) Schrad. from railroad right-of way with resistance to diuron, tebuthiuron and metribuzin. *Pest Management Science* **61**: 1035-1042.

Mengistu LW and Messersmith CG. 2002. Genetic diversity in *Kochia*. *Weed Science* **50**:498-503.

Mithila J, Hall JC, Johnson WG, Kelley KB and Riechers DE. 2011. Evolution of resistance to auxinic herbicides: historical perspectives, mechanisms of resistance, and implicatinos for broadleaf weed management in agronomic crops. *Weed Science* **59**: 445-457.

Peterson GE. 1967. The discovery and development of 2,4-D. *Agricultural History* **41**: 243-254.

Powles SB and Yu Q. 2010. Evolution in Action: Plants Resistant to Herbicides. *Annual Review of Plant Biology* **61**: 317-347.

Primiani MM, Cotterman JC and Saari LL. 1990. Resistance of kochia (*Kochia scoparia*) to sulfonylurea and imidazolinone herbicides. *Weed Technology* **4**: 169-172.

Shaner DL. 2014. Lessons learned from the history of herbicide resistance. *Weed Science* **62**: 427-431.

Smith AE and Secoy DM. 1976. Early chemical control of weeds in Europe. *Weed Science* **24**: 594-597.

Stahlman PW, Godar AS, Westera P and Brachtenbach DA. 2013. Distribution and management of glyphosate-resistant kochia (*Kochia scoparia* L. Schrad.) in the Central Great Plains of the United States, p. 31. In: *Proceedings Global Herbicide Resistance Challenge Conference*, Fremantle, Western Australia. p 31.

Stallings GP, Thill DC, Mallory-Smith CA and Shaffii B. 1995. Pollen-mediated gene flow of sulfonylurea-resistant kochia (*Kochia scoparia*). *Weed Science* **43**: 95-102.

Timmons FL. 1970. A history of weed control in the United States and Canada. *Weed Science* **18**: 294-306.

Varanasi VK, Godar AS, Currie RS, Dille JA, Thompson CR, Stahlman PW and Jugulam M. 2015. Field evolved resistance to four modes of action of herbicides in a single kochia (*Kochia scoparia* L. Schrad.) population. *Pest Management Science* **71**: 1207-1212.

Waite, J, Thompson CR, Peterson, DE, Currie RS, Olson BLS, Stahlman PW and Al-Khatib, K. 2013. Differential kochia (*Kochia scoparia*) population response to glyphosate. *Weed Science* **61**: 193-200.

Westra P, Wilson RG, Miller SD, Stahlman PW, Wicks GA, Chapman PL, Withrow J, Legg D, Alford C and Gaines TA. 2008. Weed population dynamics after six years under glyphosate- and conventional herbicide-based weed control strategies. *Crop Science* **48**: 1170-1177.

Wilson RG, Miller SD, Westra P, Kniss AR, Stahlman PW, Wicks GW and Kachman SD. 2007. Glyphosate-induced weed shifts in glyphosate-resistant corn or a rotation of glyphosate-resistant corn, sugarbeet, and spring wheat. *Weed Technology* **21**: 900-909.