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Identification of a *psbA* Mutation (Valine$_{219}$ to Isoleucine) in Powell Amaranth (*Amaranthus powellii*) Conferring Resistance to Linuron

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A Powell amaranth population suspected to be resistant (R) to linuron was discovered in a carrot field in Keswick, Ontario, Canada, in 1999. Dose–response analysis with different herbicides and DNA sequencing of the *psbA* gene encoding the D1 protein of photosystem II were done to confirm the resistance and identify its basis. A calculated resistance factor indicated a 12-fold increased resistance when linuron was applied to an R population compared with a susceptible (S) population. Moreover, the R population showed 6.4- and 3.1-fold greater resistance to two other phenylurea herbicides (diuron and monolinuron), 1.8- and 1.4-fold greater resistance to two triazine herbicides (metribuzin and prometryn), and 2.6-fold greater resistance to the triazinone metribuzin. R population was also cross-resistant to bentazon and bromoxynil when compared with S population, with a calculated resistance factor of 1.4 and 2.2, respectively. The partial nucleotide sequence of the *psbA* gene of R populations differed at two locations when compared with S populations. The first mutation coded for a Val$_{219}$Ile substitution in the deduced amino acid sequence of the D1 protein, and the second mutation was silent and encoded for a proline at position 279 in both R and S populations. The Val$_{219}$Ile substitution in the *psbA* gene is most likely the cause of this Powell amaranth population resistance to linuron and other PSII inhibitors. This is the first recorded instance of a Val$_{219}$Ile substitution in an *Amaranthus* species.

Nomenclature: Linuron; green pigweed (Canada), Powell amaranth (USA), *Amaranthus powellii* S. Wats.; carrot, *Daucus carota* L.

Key words: Herbicide resistance, mutation, PSII inhibitors, target site.

The annual weed commonly known as green pigweed in Canada and Powell amaranth in the United States is one of the major weeds affecting vegetable crops and row crops throughout North America (Costea et al. 2004). In carrots, the control of Powell amaranth relies almost exclusively on the use of herbicides that target photosystem II (PSII) (OMAFRA 2014). PSII-inhibiting herbicides act by competing with the native plastoquinone for binding at the Q_B site of the D1 protein in the PSII complex. The D1 protein is a subunit of the PSII core complex and is encoded by the chloroplast *psbA* gene. PSII inhibitors possess a greater affinity for the Q_B binding site than the plastoquinone itself, and this interrupts the normal electron transport to the cytochrome b_6f complex (Bowyer et al. 1991).

In view of their wide usage and high efficacy, PSII inhibitors exert a strong selection pressure for the emergence of resistant weed populations. Resistance to PSII-inhibiting herbicides in weeds was first reported in 1968 as a result of the constant use of triazines in crops (Ryan 1970). Since then, more than 72 plant species have developed resistance to triazines (Weed Science Society of America [WSSA] group 5) and 25 to phenylureas (WSSA group 7) as reported by the International Survey of Herbicide-Resistant Weeds (Heap 2014). In fact, PSII resistance comes second, in terms of number of weed species affected, after resistance to acetolactate synthase inhibitors. Of the species that are resistant to PSII inhibitors, the majority has been selected by the triazines (LeBaron 2008).

Resistance to PSII inhibitors affects a large number of weeds and herbicides. Most cases of plant resistance to photosynthetic inhibitors are the result of a particular mutation that modifies the target site of the D1 protein in the PSII core complex. Detailed studies done on algae and cyanobacteria have shown several possible amino acid substitutions conferring resistance to PSII inhibitors (Oettmeier 1999). These substitutions were all located at the Q_B site of the D1 protein between the amino acid phenylalanine at position 211 (Phe$_{211}$) and leucine at position 275 (Leu$_{275}$). In higher plants, eight different D1 variants have been identified so far: Leu$_{218}$ to Val in common lambsquarters (*Chenopodium album* L.) (Thiel and Varrelmann 2014); Val$_{219}$ to Ile in annual bluegrass (*Poa annua* L.) (Mengistu et al. 2000) and kochia (*Kochia scoparia* (L.) Schrad.)

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(Mengistu et al. 2005); Ala_{251} to Val in common lambsquarters (Mechant and Bulcke 2006; Mechant et al. 2008) and redroot pigweed (*Amaranthus retroflexus* L.) (Park and Mallory-Smith 2006b); Phe_{255} to Ile in shepherd's-purse (*Capsella bursa-pastoris* (L.) Medik.) (Perez-Jones et al. 2009); Phe_{255} to Val in red sorrel (*Rumex acetosella* L.) (Li et al. 2014); Ser_{264} to Gly in numerous species (Gronwald 1994); Ser_{264} to Thr in common purslane (*Portulaca oleracea* L.) (Masabni and Zandstra 1999a,b); and Asn_{266} to Thr in common groundsel (*Senecio vulgaris* L.) (Park and Mallory-Smith 2006a). All of these mutations were identified in weed populations that became resistant under selection pressure from the repetitive use of PSII inhibitors.

Linuron, a phenylurea herbicide (WSSA group 7), is often used in PRE or POST application to control annual and perennial dicotyledonous and grass weeds. Linuron is the most important if not the sole herbicide able to provide selective control of dicotyledonous weeds in carrots (OMAFRA 2014). In 1999, a carrot grower reported his lack of success with linuron to control a Powell amaranth population in his field located near Keswick, Ontario, Canada. The field had likely been subjected to many years of linuron applications (Leanne Carter, DuPont Canada, personal communication). The potential development of resistance to linuron in carrot production could have a significant effect on profitability and sustainability of this crop. Because there is no other herbicide alternative as efficient as linuron, the recourse to hand weeding could represent a significant additional cost.

The aims of this study were therefore to confirm the extent of resistance of this Powell amaranth population to linuron, determine the pattern of cross-resistance to other PSII inhibitors, and assess whether the resistance in this population of Powell amaranth from Ontario is due to modifications in the *psbA* gene and, if so, identify the specific mutations present.

Materials and Methods

Collection of Powell Amaranth Seeds. Patches of Powell amaranth that appeared to have survived linuron treatment were identified in a carrot field near Keswick in 1999. Seeds were collected from patches of the suspected resistant (R) population and from a cluster of plants at a nearby (~ 25 km) location where linuron had never been sprayed. The seeds from the unsprayed population were used as the susceptible (S) reference. Seeds from the R and S populations were planted and kept apart in a growth room. At maturity, seed heads were dried at 40 C for 48 h and cleaned using a series of screens and forced air. After cleaning, seeds were stored dry at 5 C until further use.

Seed Germination. Optimal germination was achieved by scarifying seeds with concentrated sulfuric acid (H_2SO_4, 96 to 98% by volume) for 30 s and rinsing the seeds with water, followed by a rinse with a 0.1 M solution of sodium bicarbonate and a final water rinse. Seeds were allowed to dry overnight at 25 C then stored at 5 C until planting. Germination was further enhanced by heat treatment, whereby acid-treated seeds were placed in petri dishes containing 0.6% (wt/v) agar. They were then subjected to a temperature cycle of 40 C for 16 h followed by 15 C for 2 h and 40 C for 6 h in the dark in a growth cabinet. After this heat treatment, seeds were removed from the growth cabinet and placed in a growth room where they were left to germinate for 2 to 3 d. Conditions in the growth room included a 16-h photophase at 26 C and an 8 h scotophase at 22 C. Light was provided by a mixture of incandescent bulbs and fluorescent tubes for a photosynthetically active radiation intensity of 450 $\mu E\ m^{-2}\ s^{-1}$.

Whole-Plant Dose Response. Seedlings were transplanted in 15 × 15-cm pots filled with a commercial soil mix of peat moss, perlite, dolomitic limestone, gypsum, and wetting agent (Sunshine LA4 MIX Aggregate Plus, Sun Gro Horticulture Canada, Seba Beach, Alberta, Canada). Five seedlings per pot were placed under the same growth conditions as described above. Plants were watered daily and fertilized weekly using a solution of 1.5 g L^{-1} of 20 : 20 : 20 (N : P : K) fertilizer. At the four-leaf stage, plants were sprayed with the herbicides linuron (50 to 3,000 g ai ha^{-1}), monolinuron (50 to 2,000 g ai ha^{-1}), diuron (25 to 2,500 g ai ha^{-1}), atrazine (10 to 1,000 g ai ha^{-1}), prometryn (25 to 1,000 g ai ha^{-1}), metribuzin (25 to 1,000 g ai ha^{-1}), bentazon (200 to 1,200 g ai ha^{-1}), and bromoxynil (50 to 350 g ai ha^{-1}) and compared with an untreated control. Herbicide applications were made in a laboratory chamber equipped with a sprayer calibrated to deliver 210 L ha^{-1} of herbicide solution at 275.5 kPa using compressed air and a Teejet SS8002E spray tip (Spraying Systems Co., Carol Stream, IL). All plant material was harvested at soil level 10 d after treatment and dried at 80 C for 24 h to a constant weight before recording

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biomass. The experimental design was a randomized complete block with four replications. The experiments were repeated in time.

Data Collection and Analysis. For the S and R populations, results were expressed on a dry biomass basis as a percentage of the untreated control. The data were subjected to ANOVA using PROC GLM in SAS (version 9.3, SAS Institute, Cary, NC). Data were pooled before dose–response analysis because no significant interaction between treatments and replications was found. Dose–response curves expressing dry weight were generated using PROC NLIN in SAS. The dose required to reduce plant dry weight by 50% (GR_{50} values) for each population were predicted using the log-logistic equation described by Seefeldt et al. (1995) with the following equation relating the dry weight y to the herbicide dose x,

$$y = C + \frac{D - C}{1 + (x/\mathrm{GR}_{50})^{b}} \quad [1]$$

where C represents the lower limit, D the upper limit, and b the slope at GR_{50}. Resistance factors were calculated by dividing GR_{50} values of the R biotype by the GR_{50} values of the S population.

Molecular Analysis. Several samples of young leaf tissue (100 mg) were taken from untreated R and S Powell amaranth plants grown in greenhouses. They were freeze-dried and stored at −80 C until further use. Once plant tissues were removed from the freezer, they were placed in a prechilled mortar filled with liquid nitrogen and ground with a pestle into fine powder. The DNA was extracted from the ground leaf tissue according to the FastDNA protocol (FastDNA KIT, BIO 101, MP Biomedicals, Santa Ana, CA), except with centrifugation at 4 C for 10 min. DNA samples were diluted 10-fold with distilled deionized water to an approximate concentration of 10 ng μl^{-1} and returned to the freezer before further analysis.

A 1,050-bp DNA fragment of the *psbA* gene encoding the herbicide-binding niche of the D1 protein was amplified by polymerase chain reaction (PCR) using two pairs of primers (Diebold et al. 2003). The first set of primer sequences, 5′-TGTGCTTGGGAGT CCCTG-3′ and 5′-AGGCATACCATCAGAAAA-3′, amplified the first 527 bases of the *psbA* gene. The second primer set, 5′-TGTTGCATATTCAGCTCC-3′ and 5′-TTGTAGATGGAGCTTC(AG)A(CT)AG-3′, amplified 583 bases of the second half. Approximately 50 bp were allowed for overlap of the two halves to permit proper sequence alignment. Primers were constructed using a DNA synthesizer (ABI DNA Synthesizer Model 394, Applied Biosystems Inc., Foster City, CA).

Amplification reactions contained 50 ng of total DNA, 900 nM of each primer, 50 mM of $MgCl_2$, 200 µM of each dNTP, 1.25 units of Platinum *Taq* DNA polymerase (Life Technologies Inc., Burlington, Ontario) and a 1× concentration of supplied buffer in a final volume of 50 µl. The reaction mixtures were subjected to a 3-min incubation at 97 C; 40 cycles of 0.5 min at 97 C, 1 min at 58 C, and 1.5 min at 72 C; then 8 min at 72 C in a RoboCycler 96 temperature cycler (Stratagene, La Jolla, CA). One tenth of the PCR products were separated by electrophoresis on a 1% (wt/v) agarose gel run with TBE (Tris base + boric acid + EDTA) buffer. Ethidium bromide was added to the gel mixture at a concentration of 5 µl for every 100 µl of gel solution.

For sequencing, *psbA* amplified DNA fragments were extracted from the remaining PCR products using Amicon or Microcon PCR centrifugal filter devices according to manufacturer procedures (EMD Millipore Canada, Etobicoke, Ontario). Once purified, DNA samples were submitted to Guelph Molecular Supercentre (Laboratory Service Division, University of Guelph, Guelph, Ontario) for sequencing. Multiple sequences were obtained for each sample in both forward and reverse directions to minimize sequencing errors. The sequences for S plants were analyzed first and compared with R sequences to determine whether a nucleotide change occurred using ClustalW software (ClustalW multiple sequence alignment tool, European Bioinformatics Institute, available online: http://www.ebi.ac.uk/clustalw/). Gene Runner (http://www.generunner.net/) was used to obtain the complementary strand of the sequenced *psbA* gene fragments, and the ExPASy translate tool (ExPASy: SIB bioinformatics resource portal, http://www.expasy.org) was used to determine the peptide sequence.

Results

Whole-Plant Dose Response. Dose–response analysis confirmed that the R population is resistant to linuron. GR_{50} values of R and S populations did not overlap at the 95% confidence interval, indicating that the dose–response curves were significantly different (Table 1). Compared with the S population, R had a 11.6-fold resistance level based on GR_{50} values.

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Table 1. Parameters (95% confidence intervals) of the log-logistic equations for the whole-plant dose response of resistant (R) and susceptible (S) populations and resistance factors for the eight herbicides tested.[a,b]

Herbicide	Population	*D* g db	*C* g db	*B* g ai	GR_{50}	RF
			g ai^{-1}			
Linuron	R	99.9 (99.5–100.3)	1.71 (1.6–1.8)	1.06 (0.98–1.13)	112.6 (104.0–121.2)	11.6
	S	99.9 (99.5–100.3)	1.71 (1.6–1.8)	1.03 (0.95–1.12)	9.7 (9.0–10.5)	
Diuron	R	101.3 (99.8–101.8)	0.88 (0.8–0.9)	1.50 (1.40–1.59)	157.4 (148.7–166.14)	6.4
	S	101.3 (99.8–101.8)	0.88 (0.8–0.9)	5.6 (3.5–7.7)	24.5 (23.8–25.1)	
Monolinuron	R	98.4 (91.5–105.3)	2.24 (2.1–2.4)	0.88 (0.82–0.94)	193.0 (174.10–211.79)	3.1
	S	98.4 (91.5–105.3)	2.24 (2.1–2.4)	1.0 (0.9–1.2)	62.5 (56.3–68.6)	
Atrazine	R	101.3 (94.2–108.4)	0.01 (−0.05–0.06)	1.14 (1.01–1.27)	37.87 (33.54–42.20)	1.8
	S	101.3 (94.2–108.4)	0.01 (−0.05–0.06)	1.7 (1.5–1.9)	20.9 (19.1–22.8)	
Metribuzin	R	99.4 (92.4–106.4)	1.29 (1.2–1.4)	0.76 (0.72–0.80)	67.78 (63.33–72.23)	2.6
	S	99.4 (92.4–106.4)	1.29 (1.2–1.4)	1.6 (1.5–1.7)	26.1 (24.9–27.3)	
Prometryn	R	99.7 (92.7–106.7)	0.72 (0.7–0.8)	0.99 (0.92–1.08)	37.5 (33.85–41.11)	1.4
	S	99.7 (92.7–106.7)	0.72 (0.7–0.8)	1.3 (1.2–1.4)	26.4 (24.2–28.7)	
Bentazon	R	99.8 (91.8–105.6)	0.88 (0.8–0.9)	0.99 (0.91–1.06)	37.61 (34.05–41.17)	1.4
	S	99.8 (91.8–105.6)	0.88 (0.8–0.9)	1.3 (1.2–1.4)	26.5 (24.3–28.7)	
Bromoxynil	R	98.7 (92.8–106.8)	0.46 (0.4–0.5)	2.01 (1.81–2.21)	106.55 (100.23–112.87)	2.2
	S	98.7 (92.8–106.8)	0.46 (0.4–0.5)	1.9 (1.6–2.2)	48.7 (45.5–51.9)	

[a] Data are pooled from two separate experiments for each herbicide.
[b] Abbreviations: db, dry biomass; RF, resistance factor calculated as the ratio of GR_{50} values for the R biotypes over S biotypes.

As with linuron, R populations also showed resistance to other phenylurea herbicides such as diuron and monolinuron (Table 1). Resistance factors were 6.4 for diuron and 3.1 for monolinuron. Herbicides from the triazine and triazinone groups (WSSA group 5) were also tested on R and S population. Compared with the S biotype, R also exhibited resistance to the triazines, with resistance factors of 1.8 for atrazine and 1.4 for prometryn. Resistance to the triazinone metribuzin was also observed with a resistance factor of 2.6.

Cross-resistance to PSII inhibitors from other chemical families was also observed (Table 1). Dose–response results for the R population were different from the S population after treatment with the benzothiadiazole, bentazon, and the nitrile, bromoxynil (both in WSSA group 6). GR_{50} values were different between R and S biotypes after bentazon application, with a resistance factor of 1.4. The R population was also different from S after bromoxynil application with a resistance factor of 2.2.

Genetic Analysis. Partial DNA sequences of the *psbA* gene encoding for the D1 protein were obtained from the Powell amaranth S and R populations. The *psbA* DNA fragment from the S biotype was compared with the known smooth pigweed (*Amaranthus hybridus* L.) sequence (GenBank accession K01200.1 GI: 33605), and both sequences were perfectly matched.

The *psbA* nucleotide sequence from the R population differed, however, at two locations when compared with the sequence of the S population (Table 2). A transition mutation (G to A) at position 655 of the DNA sequence resulted in an amino acid substitution of valine to isoleucine at position 219 in the D1 protein. The second mutation was a transversion from G to T at position 837 and was silent at the protein level, because it resulted in a proline at position 279 for both R and S populations. The $Val_{219}Ile$ substitution in the D1 protein most likely explains resistance to linuron and other PSII inhibitors in the resistant Powell amaranth population.

Discussion

Analysis of the dose–response data confirmed resistance to linuron in a population of Powell amaranth from southern Ontario. Additionally, this population showed cross-resistance to the phenylurea herbicides diuron and monolinuron. A lower level of resistance was also observed with other PSII inhibitors, including the triazines atrazine and prometryn; the triazinone metribuzin; the nitrile bromoxynil; and the benzothiadiazole bentazon.

The mutation in the R population of *Powell amaranth* is the same reported for *Chlamydomonas* and *Synechococcus*, after site-directed mutagenesis, and in cell cultures of red goosefoot (*Chenopodium rubrum* L.) (Oettmeier 1999). In theses organisms, varying levels of cross-resistance and negative cross-resistance were observed. For example, compared with the respective wild type, the mutants were 200-fold resistant to

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Table 2. Nucleotide and amino acid polymorphisms inferred from alignment of *psbA* sequences from three individuals of each population.

	Polymorphism			
	1		2	
	Nucleotide[b]	Amino acid	Nucleotide	Amino acid
Population[a]	655–657	219	835–837	279
S	GTA	Val	CCG	Pro
R	ATA	Ile	CCT	Pro

[a] Abbreviations: S, susceptible; R, resistant.

[b] Nucleotide numbers designate codon position of the sequence in which the polymorphism was found. Polymorphisms within codons are underlined.

metribuzin, 50-fold resistant to ioxynil, and 15- to 32-fold resistant to diuron. In contrast, supersensitivity (negative cross-resistance) was observed with some triazinones other than metribuzin. In resistant Powell amaranth, no supersensitivity to the herbicides tested was observed.

The pattern of resistance observed for phenylureas and triazines suggested that the cause of resistance was probably different from the $Ser_{264}Gly$ substitution frequently observed in the case of triazine-selected resistance to PSII (Powles and Yu 2010). The resistance profile revealed by the R population was very similar to that found in PSII-resistant populations of annual bluegrass and kochia (Mengistu et al. 2000, 2005). In these two species, a similar amino acid substitution at position 219 in the D1 protein was associated with resistance to metribuzin, diuron, and tebuthiuron. However in resistant kochia, this mutation made the plants more susceptible to bromoxynil, even at 50% of the recommended rate. This is different from what we observed in our R population showing resistance to bromoxynil herbicides. Similar mutations in different organisms may show different ranges of cross-resistance to the same herbicide, perhaps because of other mechanisms at play in the plant, such as different rates of absorption, altered translocation, or increased metabolism.

In several weed species, herbicide resistance to PSII is often associated with a mutation at residue 264 in the D1 protein of the PSII complex. This mutation has been shown to cause inhibition of the electron transport system, leading mutated plants to be less fit than their susceptible counterparts (Gronwald 1994). However, in the case of mutation 219, no effect on the electron transfer system of PSII in *Chlamydomonas reinhardtii* P. A. Dang was observed (Erickson et al. 1985). This could also be the case with the R plants in our study.

Previous studies with organisms having the $Val_{219}Ile$ mutation have shown resistance to diuron, metribuzin, hexazinone, bromoxynil, and atrazine (Mengistu et al. 2000, 2005; Oettmeier 1999). Our study determined that this mutation also confers resistance to linuron, monolinuron, prometryn, and bentazon, clearly showing that this alteration in the D1 protein confers a very broad spectrum of cross-resistance that has implications for potential management of weeds. Although the population under study was likely selected by a single herbicide (linuron), it also possesses cross-resistance to all the PSII inhibitors tested. Consequently, although the level of resistance to some of those inhibitors is low, it is still enough to prevent them from providing adequate control. As a result, this whole class of herbicides is practically rendered ineffective.

$Val_{219}Ile$ Powell amaranth mutants could become an important problem to growers. In the field, their presence would greatly diminish the choice of several alternative herbicides. Although resistant Powell amaranth plants can be damaged after a linuron application (data not shown), they recover fast in a noncompetitive crop and are eventually competitive enough with the crop to produce seeds that will enrich the seed bank. The choice of herbicides that can be used in POST application in carrots is limited. Linuron and prometryn are the only two herbicides that are registered for this use in Ontario (OMAFRA 2014), which is why growers use these year after year. However, the use of prometryn would not be advisable, because it would likely fail to provide adequate control on Powell amaranth mutants selected by linuron.

The spread of resistant weeds after their initial appearance is very often a concern. Gene flow through pollen or seed dispersal can accelerate selection for resistance in areas adjacent to those in which the initial selection occurred (Maxwell and Mortimer 1994). In the current case, gene flow through pollen is not a concern because the gene conferring resistance is located within the plastome, thereby ensuring maternal inheritance. The possibility of gene flow through seed dispersal is, however. present because *Amaranthus* species produce a high number of very small seeds that are easily transported through natural or artificial means (Costea et al. 2004). Growers would therefore be advised to ensure sanitation of machinery and equipment to avoid spreading linuron-resistant populations.

To avoid further selection of linuron resistance, one option could be crop rotations that allow the use of herbicides with different modes of action.

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Herbicides should be used within Integrated Weed Management (IWM) strategies to diminish any risks of herbicide resistance. Under IWM strategies, weed populations are not exposed to the same selection pressure repeatedly, which would likely limit the likelihood of linuron resistance spreading further. However, if growers continue to rely heavily on a single weed management tool, the problem likely will continue to expand.

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