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RESEARCH ARTICLE

Detection of ALS gene mutations conferring pyroxsulam resistance in wild mustard (*Sinapis arvensis* L.) from Kurdistan, Iraq

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Abstract

Wild mustard is a diploid (2n = 18) annual winter weed that can reduce crop yields by between 48 % and 71 %. This weed is nowadays resistant to acetolactate synthase and even to new herbicides. In an experiment with wild mustard with suspected resistance to Pyroxsulam herbicide, seeds were collected and planted in a pot in a growth chamber. Five concentrations of Pyroxsulam herbicide were applied when the wild mustard had developed 2 to 3 leaves. After 2 weeks of herbicide application, 2 leaves sterilised with 70 % ethanol were sampled. Sequence analysis of the ALS gene detected twenty target site mutations in ORF1 and ORF3 with amino acid substitutions. ALS gene mutations were detected at multiple codons, with the highest mutation frequency at 1.08 % Pyroxsulam concentration. Significant differences between concentrations used in the study and single-nucleotide polymorphisms were found in ORF1, ORF3 and combinations of the two fragments. The herbicide concentrations were indicated to play a significant role in nucleotide substitutions in wild mustard. This herbicide dose-response experiment confirmed that wild mustard resisted the Pyroxsulam herbicide. This is the first report of such mutations in wild mustard from Kurdistan, Iraq.

Keywords: ALS gene; mutation; pallas OD; resistance; *Sinapis arvensis*

Abbreviations: Ala: Alanine acid; ALS: Acetolactate synthase; Arg: Arginine acid; Asp: Asparagine acid; Asp: Asparagine acid; Asp: Aspartic acid; BLAST: Basic local alignment search tool; Cys: Cysteine acid; DNA: Deoxyribonucleic acid; Gln: Glutamine acid; Glu: Glutamic acid; Gly: Glycine acid; His: Histidine acid; Ile: Isoleucine acid; Leu: Leucine acid; NCBI: The National center for biotechnology information; ORF: Open reading frame; Pallas OD: Pallas oil dispersion; PCR: Polymerase chain reaction; Phe: Phenylalanine acid; Pro: Proline acid; SEM: Standard error of the mean; Ser: Serine acid; SNP: Single Single-nucleotide polymorphism; Thr: Threonine acid; Trp: Tryptophan acid; Tyr: Tyrosine acid; Val: Valine acid

Introduction

Wild mustard (*Sinapis arvensis* L.) is a diploid (2n=18) annual winter weed distributed in temperate regions around the world and is commonly found in winter cereals, pulses and oilseed crops (1, 2). The plant is considered a noxious weed due to its high seed production, which is about 3500 seeds per plant annually and is readily dispersed in soil to be a massive seed bank. The weed can reduce crop yields by between 48 % and 71 % in severe competition (3, 4). However, differences in relative abundance among Brassicaceae weed species might be attributed to the length of time from emergence, adaptability and their competitive ability against crops and other weeds (5). Chemical weed control is considered a sufficient method for weed management because it is approachable, less time-consuming as well and economical and several herbicides have been applied in crop fields to control the weeds efficiently.

These include sulfosulfuron, chlorsulfuron, rimsulfuron, metsulfuron, 2,4-D and dicamba (6, 7). Recently, a new herbicide has been introduced and applied in an attempt to eradicate the weeds in wheat fields. This is Pyroxsulam (Pallas OD), an herbicide which belongs to the sulfonylurea group and is responsible for inhibiting the acetolactate synthase (ALS) (8). On the other hand, weed resistance to herbicide application can pose problems in weed management. However, the first report of wild mustard resistance to ALS-inhibiting herbicides was documented in Canada and the USA in 1982 and 1983, respectively and nowadays has become a worldwide issue (2, 9). Weed resistance to herbicides can occur because the herbicide cannot bind to the target site due to a mutation. After all, the herbicide cannot bind to the target site due to intense protein synthesis in the enzyme region, or because the plant metabolizes the herbicide very rapidly and tolerates the

phytotoxic effect of the herbicide; especially, in agricultural lands where monoculture is practiced for many years, weed resistance to herbicides can result from continuous use of herbicides with the same mode of action (5). The Pallas OD herbicide in Iraq and particularly in the Kurdistan Region, faces resistance to acetolactate synthase in wild mustard, as farmers claim recovery of the weed following Pallas OD application. This study aimed to identify target-site mutations in the ALS gene responsible for Pyroxsulam resistance in wild mustard populations from Kurdistan.

Materials and Methods

Plant material

Seeds of wild mustard (*Sinapis arvensis* L.) with suspected resistance to Pyroxsulam herbicide (Pallas OD) were collected from a cereal plot of winter wheat located in a field in Qaja Village, Ranya, Kurdistan (36.192182" N, 44.727187" E). The fields were treated with the herbicide for over five consecutive years, hereafter referred to as resistant biotypes.

Herbicide dose response

The herbicide dose-response experiment was conducted in a growth chamber in the summer of 2023 at the Research Centre, Erbil Polytechnic University. The experiment was arranged in a completely randomised design with three replications. Three seeds were planted 1 cm deep in 1.5 kg pots containing sandy soil. The light and temperatures (18-25 °C) were controlled. Pots were watered daily to field capacity. When the seedlings reached the two to three-leaf stage, the Pallas OD (4.5 %) herbicide was applied in five concentrations of 0, 0.27, 0.54, 0.81 and 1.08 % (1). After two weeks of application, the leaves of the wild mustard plants were sampled.

DNA isolation and selection of primers

Two plant leaves from *Sinapis arvensis* L. per pyroxsulam herbicide concentration (plant individuals that were not shrivelled) were sterilised using ethanol 70 % and stored in a -20 °C refrigerator and lyophilised. DNA was isolated using a plant DNA extraction kit (Beta Bayern GmbH, .90453 Bayern, Germany) following the manufacturer's protocol. Two pairs of Single Nucleotide Polymorphism (SNP) DNA primers were used to amplify the conserved domain regions of the Acetolactate Synthase (ALS) gene, spanning all the known mutation sites reportedly contributing to ALS resistance (Table 1).

DNA amplification using the polymerase chain reaction (PCR)

PCR amplification was done for open reading frames 1 and 3 (ORF1 and ORF3) of ALS gene so that each PCR reaction involved a total volume of 50 µL of reaction mixture containing 25 µL of 2x Taq DNA polymerase master mix (AMPLIQON A/S Stenhuggervej 22), 4 µL of 10 Picomol (pmol) primers, 16 µl of DNase free water and 5 µL of DNA template by Bioresearch PTC-200 gradient thermocycler. The PCR profile was denatured at 95 °C for 5 min, followed by 95 °C for 30 sec. Then, a 54 °C annealing temperature was applied for 30 sec, with extension at 72 °C for 1 min. This was followed by one final extension cycle at 72 °C for 10 min and an indefinite hold at 4 °C.

ALS gene sequencing

The ALS gene fragments were amplified from each treatment and purified using the FAVORGEN DNA Purification Kit (Taiwan). These purified gene fragments were sent to Macrogen (https://dna.macrogen.com/) to be sequenced with the primers listed above. The sequencing results were visualised using Finch TV and aligned using BLASTn and Blastx software from NCBI. Alignment was performed using as a consensus sequence, the Gene Bank accession FJ655877 of *S. arvensis*.

Data analysis

Sequencing of ALS gene data was analysed using the NCBI Alignment. The nonparametric data were subjected to Dunn's multiple comparison test to determine the significance of the differences between means of the concentrations, single nucleotide polymorphism and amino acid substitutions using GraphPad Prism Software.

Results and Discussion

Sinapis arvensis L. (2n = 18) aggressively invades wheat fields as a broad-leaf weed. It is the main factor that reduces growth and causes significant yield losses in wheat production. Pyroxsulam, one of wheat's main herbicides, controls dicotyledonous weeds by inhibiting the plant enzyme acetolactate synthase, essential for synthesising branched-chain amino acids. The inhibition of amino acid production subsequently inhibits cell division and causes death in susceptible plants. Local farmers have highlighted that for more than 10 consecutive years in the area, the resistant population had been mainly treated with pyroxsulam herbicide.

Two fragments (ORF1 and ORF3) of the ALS gene were amplified and the sequence of the partial genes was determined by the BLASTn and BLASTx programs from gene bank and compared to gene bank accession FJ655877 *Sinapis arvensis* L. This was used to compare our amplified sequences with control or non-treated herbicide sequences. The results from the BLAST programs indicated that the highest query sequence was a 100 % identity with the control in ORF1 and ORF3 (Table 2-3). The nucleotide variation in ORF1 and ORF3 of ALS gene sequences varied among *Sinapis arvensis* L. treated with various concentrations. Research indicates that resistance occurs when a nucleotide difference leads to an amino acid point mutation at the binding site (10). Based on the results, ORF1 has recorded 18 nucleotide substitutions in different codons using various concentrations. The replacement of one nucleotide did not lead to amino acid substitutions at a position corresponding to only

Table 1. Single nucleotide polymorphism DNA primers used to acetolactate synthase (ALS) the gene sequence from *Sinapis arvensis* L.

Primer	Direction	Sequence 5'-3'	Amplicon size (bp)	Reference
ALS1 (ORF1)	Forward	TCARTACTWAGTGCKACCATC	639	(14)
ALS1 (ORF1)	Reverse	GGRGAAGCCATTCCTCC		
ALS2 (ORF3)	Forward	GAAGCCCTCGARCGTCAAGG	501	
ALS2 (ORF3)	Reverse	CATAGGTTGWTCCCARTTAG		

ORF = Open Reading Frame

Table 2. Detection of single-nucleotide polymorphism in ORF1 and ORF3 of the ALS gene codon numbers of *Sinapis arvensis* L. in different concentrations of pyroxsulam herbicide

Pyroxsulam herbicide concentration %	SNP of ORF1																	SNP of ORF3				
	424	425	429	430	454	455	465	471	474	484	493	495	511	518	519	532	543	44	52	64	103	157
	D	G	F	I	Q	H	R	R	T	D	A	A	D	D	T	Q	A	S	R	Y	T	P
Control	GAC	GGT	TTC	ATC	CAG	CAT	CGG	AGA	ACA ACT	GAG	GCA	GCT	GAT	GAT	GAT	ACA	CAA	ACC	CGT	TAC	ACA	CCC
0.27	GAC	GGT	TTC	ATC	CAG	CAT	CGA	AGA	ACG ACT	GAG	GCA	GCT	GAT	GAT	GAT	ACA	CAA	ACC	CGC	TAC	ACA	CCA
0.54	GAC	GGT	TTC	ATC	CAG	CAT	CGG	AGA	ACA AAT	GAT	GCA	GCT	TAT	GAT	GAT	ACA	CAT	ACC	CGT	TAC	ACA	CCA
0.81	GAC	GGT	TTC	ATC	CAG	CGT	CGG	GGA	ACA ACT	GAG	GCA	TCT	GAT	GAT	GAT	ATA	CGA	ACC	CGC	TAC	ACA	CCA
1.08	GGC	TGT	CTC	GTC	CAT	CGT	CGG	AGA	ACA ACT	GAG	GCT	GGT	GAT	GGT	GCA	CAA		AGC	CGT	GAC	GCA	CCC

Table 3. Detection of substituted and non-substituted amino acids in ORF1 and ORF3 in the ALS gene of *Sinapis arvensis* L. treated with different concentrations of pyroxsulam herbicide

Pyroxsulam herbicide concentration %	ORF1				ORF3			
	Gene name and position sequences	Nucleotide changed	Amino acid substituted	Codon number changed	Gene name and position sequences	Nucleotide changed	Amino acid substituted	Codon number changed
Control	ASL-ORF1	Wilde	Wilde type	Wilde	ASL-ORF3	Wilde	Wilde type	Wilde
0.27	ASL-ORF1	G→A	None	465	ASL-ORF3	T→C	None	52
		A→G	None	474		C→A	None	157
0.54	ASL-ORF1	G→T	E→D/ glu →asp	484	ASL-ORF3	C→A	None	157
		G→T	D→Y / asp → tyr	511				
		A→T	Q→H / gln → his	532				
		C→A	T→N / thr →asn	543				
0.81	ASL-ORF1	A→G	H→R / gln → his	455	ASL-ORF3	T→C	None	52
		A→G	R→G/ arg→gly	471		C→A	None	157
		G→T	A→S/ ala→ser	495				
		C→T	T→I/ thr→ ile	519				
		A→G	Q→R / gln → arg	532				
1.08	ASL-ORF1	A→G	D→G/ asp → gly	424	ASL-ORF3	C→G	None	44
		G→T	G→C/ gly→ cys	425		T→G	Y→ D/ tyr→asp	64
		T→C	F→ L/ phe→ leu	429		A→G	T→A/ thr→ala	103
		A→C	I→V/ ile→ val	430				
		G→T	Q→H/ gln → his	454				
		A→G	H→R / gln → his	455				
		A→T	None	493				
		C→G	A→G/ ala→ gly	495				
		A→G	D→G/ asp → gly	518				
		A→G	T→A/ thr → ala	519				

0.27 % concentration in sites 465 (CGG-CGA) and 474 (ACA-ACG) and one site in terms of 1.08 % concentration at 493 (GCA-GCT). This was assumed not to be associated with resistance to ALS inhibitors, indicating that these substitutions are neutral. However, a set of target site mutation resistances in 484 (Glu-Asp), 511 (Asp-Tyr), 532 (Gln-His) and 543 (Thr-Asn) for 0.54 % concentration, 455 (Gln-His), 471 (Arg-Gly), 495 (Ala-Ser), 519 (Thr-Ile) and 532 (Gln-Arg) for 0.81 % concentration and 424 (Asp-Gly), 425 (Gly-Cys), 429 (Phe-Leu), 430 (Ile-Val), 454 (Gln-His), 455 (Gln-His), 495 (Ala-Gly), 518 (Asp-Gly) and 519 (Thr-Ala) for 1.08 % concentration have been substituted amino acids, suggesting that these sites are highly variable. Previous studies have shown that different mutations in different sites in the ALS gene are responsible for the resistance of *Sinapis arvensis* L. to ALS inhibitors (1, 11). On the other hand, in ORF3, two out of eight nucleotide substitutions led to amino acid substitutions resulting from 1.08 % concentrations at codons 64 (Tyr-Asp) and 103 (Thr-Ala). This indicated that only these two substituting amino acids correlated with resistance to ALS inhibitors at high levels of pyroxsulam herbicide in ORF3. However, other substitutions are all neutral. Research indicates that the channel that leads to the ALS active site is blocked by ALS-inhibiting herbicides. Differences in binding efficacy are translated by differences in herbicide compound structure (12). These differences have implications for amino acid substitutions that confer resistance. The present study suggests that the resistance mechanism involving pyroxsulam is at least one or more than one factor of factors detailed below: the frequent use of this herbicide, how they have been used, low herbicide absorption or translocation and the high selection pressure it exerts (13, 14).

The ALS-inhibitor resistance noted in this study (Fig. 1-2) appeared commonly due to target site resistance, specifically single-nucleotide polymorphisms in the ALS gene sequence, which result in amino acid substitution (15). Weeds such as *Sinapis arvensis* L. have also been reported to be resistant to ALS inhibitors through non-target-site resistance, which occurs through increased herbicide metabolism that detoxifies the herbicide to sub-lethal concentrations within the plant (15). The challenge of managing herbicide resistance is further complicated by accumulating both resistance mechanisms within a single species biotype (16, 17). Therefore, developing new herbicide modes of action does not seem viable for controlling herbicide-resistant weeds (18, 19).

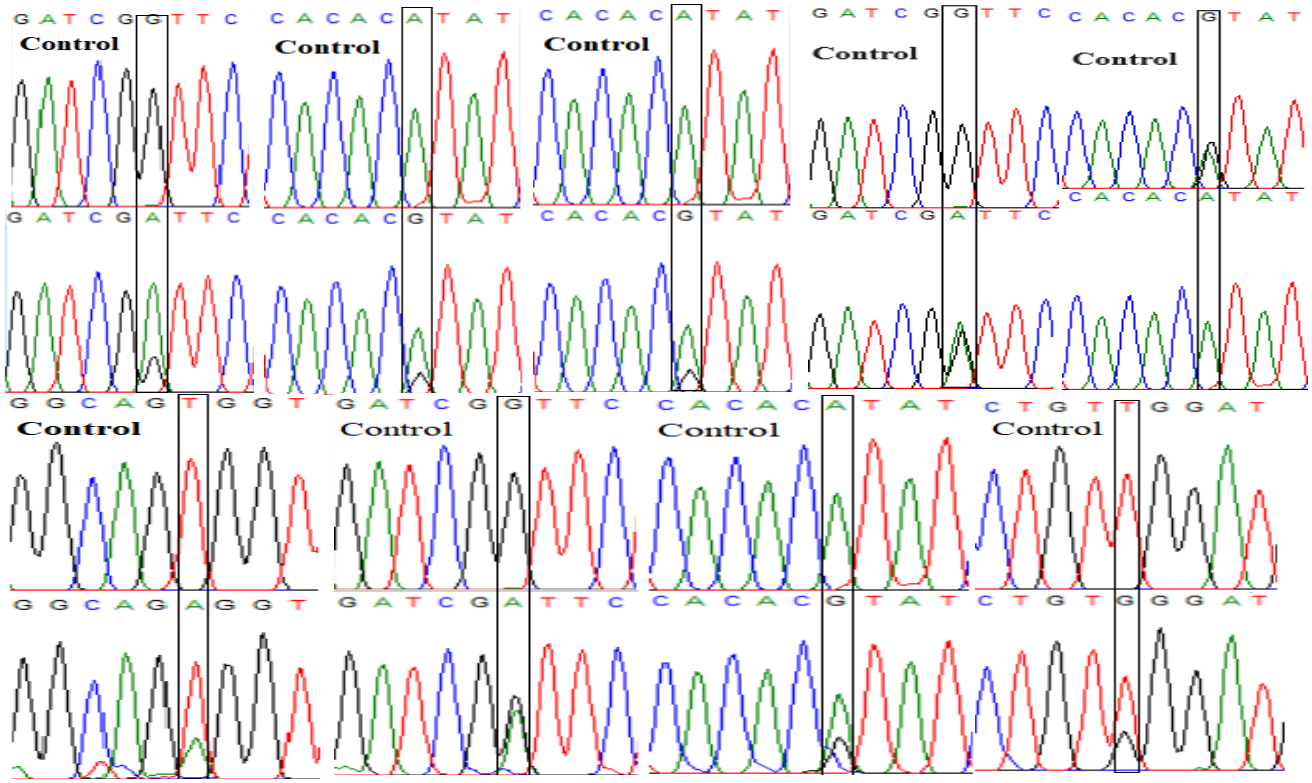

Fig. 1. Single-nucleotide polymorphisms of ORF1 from ALS gene sequences of *Sinapis arvensis* L. treated with various concentrations of pyroxsulam herbicide.

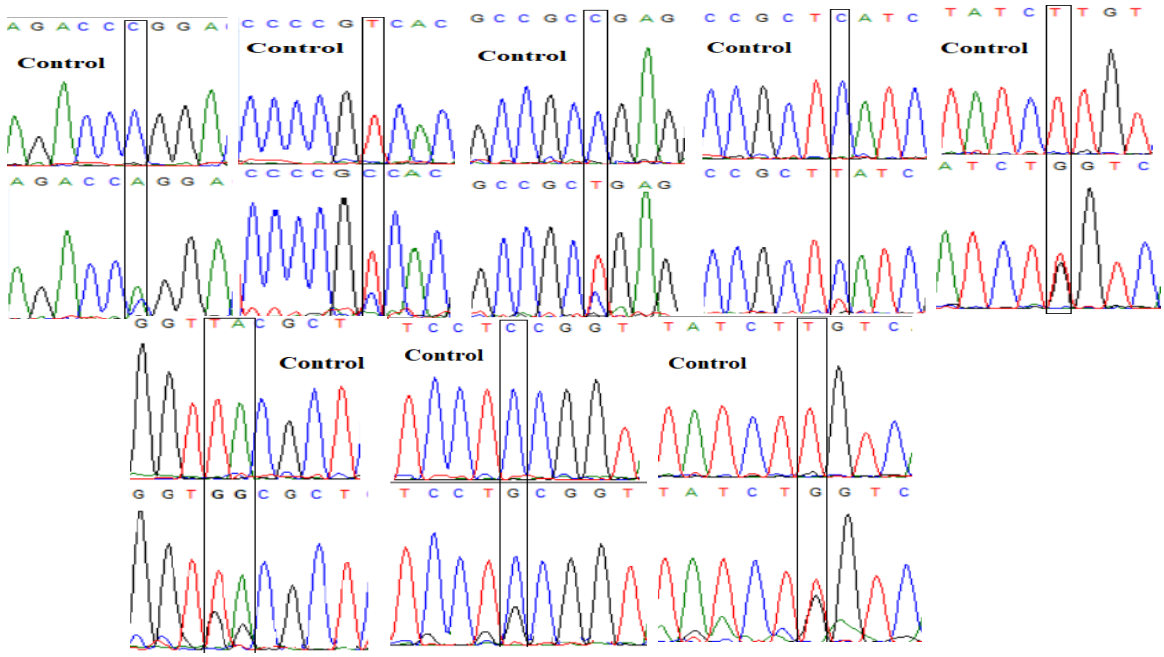

Fig. 2. Single-nucleotide polymorphisms of ORF3 from ALS gene sequences of *Sinapis arvensis* L. treated with various concentrations of pyroxsulam herbicide.

The role of the mutations in ALS genes expressing herbicide resistance was determined by comparing concentrations, single-nucleotide polymorphisms and amino acid substitutions to verify the substitution of amino acids. Significant differences between concentrations used in the study and single-nucleotide polymorphisms were found in ORF1, ORF3 and in combinations of the two fragments (ORF1 and ORF3) in the ALS gene in *Sinapis arvensis* L. (Fig. 3-5). This indicated that the herbicide concentrations have a significant role to play in nucleotide substitutions in *Sinapis arvensis* L. Acetolactate-synthase inhibitor could be taken up quickly through the roots and leaves (20). To reduce the risk of herbicide resistance, the 13 best management practices can be utilised by the grower. Two out of the 13 key suggestions for growers stated the need to change weed management practices and use multiple herbicide sites of action. Applying numerous herbicide sites of action includes the application of various multiple herbicide sites of action in sequence throughout the growing season and utilising combinations of numerous herbicide sites of action groups. Research indicates that this strategy delays the evolution of weed resistance by reducing selection pressure from one site of action and decreasing the probability that resistant individuals within weed populations reproduce and remain alive (21). Similarly, herbicide tank mixtures are generally more effective at delaying herbicide resistance than annual herbicide rotations (22).

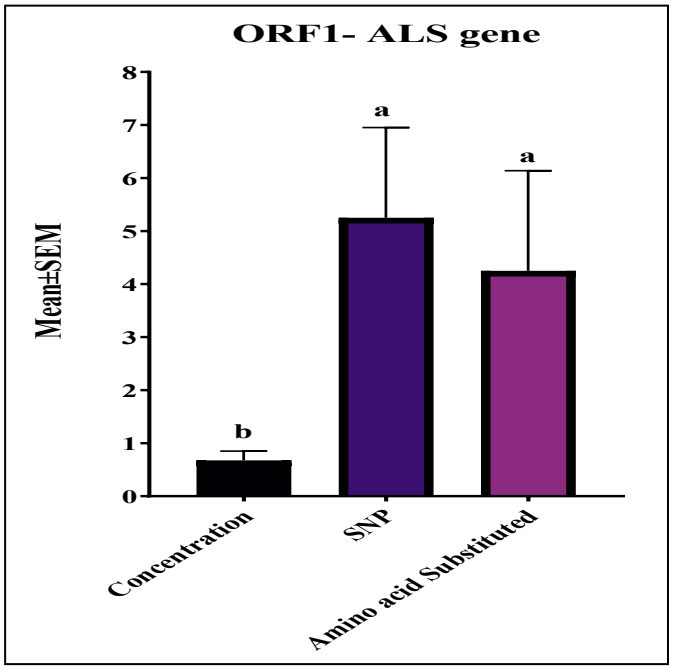

Fig. 3. Mean of concentration, single nucleotide polymorphism and amino acid substitution in *Sinapis arvensis* L. in ORF1 in the ALS gene.

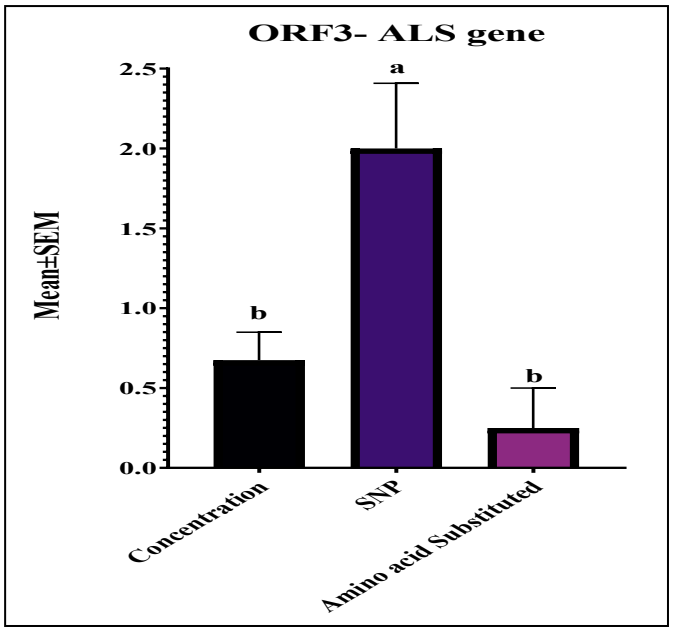

Fig. 4. Mean of concentration, single nucleotide polymorphism and amino acid substitution in *Sinapis arvensis* L. in ORF3 in the ALS gene.

Conclusion

Twenty target site mutations in ORF1 and ORF3 in different codons in the ALS gene sequence in *Sinapis arvensis* L. treated with various doses of concentrations of Pyroxsulam herbicide have been detected. This is a result of the substitution of amino acids, leading to the plant's resistance against this herbicide in the field. ALS is a nuclear gene that is normally inherited, even though it functions in plastids. Thus, pollen and seed both spread resistance ALS alleles (23). Consistently, a high magnitude difference between concentration and single-nucleotide polymorphism in ORF1 and ORF3 and a combination of ORF1 and ORF3, revealed resistance of the plant against the herbicide. Therefore, this study demonstrated that Sinapis arvensis L. developed high-level resistance to different concentrations of pyroxsulam herbicide. Management, new herbicides and new mechanisms for growers are essential for controlling *Sinapis arvensis* L.

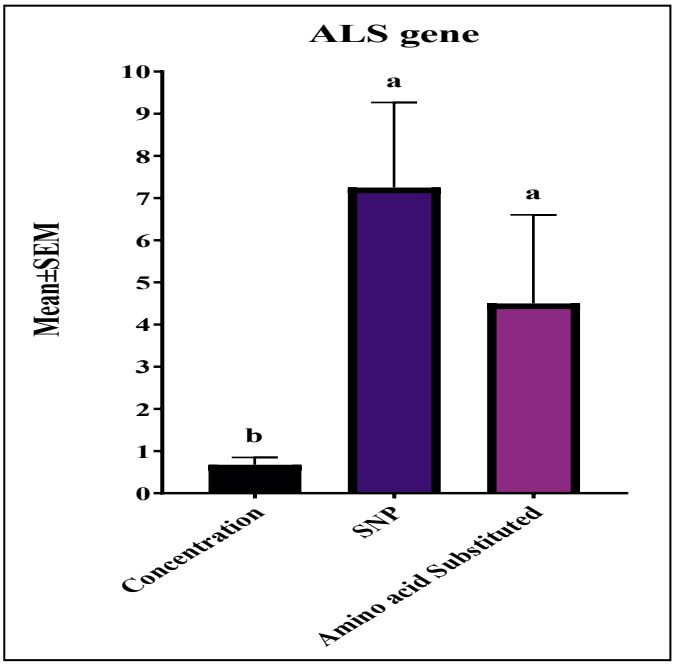

Fig. 5. Mean of concentration, single nucleotide polymorphism and amino acid substitution in *Sinapis arvensis* L. in combination with ORF1 and ORF3 in the ALS gene.

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Authors' contributions

NRM and IAS conducted the planting seed in the chamber growth, leaf sampling, writing and the article. MAA carried out DNA extraction and sequence alignment and data analysis. All authors read and approved the final version of the manuscript.

Compliance with ethical standards

Conflict of interest: Authors do not have any conflict of interests to declare.

Ethical issues: None

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