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Original Article

Resistance of Egyptian Field Populations of *Spodoptera littoralis* (Lepidoptera: Noctuidae) to Emamectin Benzoate and Role of Detoxification Enzymes

Seham Mansour Ismail*

Insect Population Toxicology Department, Central Agricultural Pesticides Laboratory, Agriculture Research Center, 12618, Giza, Egypt

*Corresponding Author E-mail: Seham.Ismail@arc.sci.eg

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ABSTRACT

Background: One of the most significant pests that harm numerous agricultural crops is the cotton leafworm *Spodoptera littoralis* (Boisduval) and it has evolved resistance to various insecticides across most chemical classes, including the novel insecticide emamectin benzoate.

Methods: The sensitivity of emamectin benzoate was evaluated in Egyptian cotton leafworm field populations.

Results: According to bioassays performed using the leaf-dipping method, emamectin benzoate sensitivity varied among 5 field populations, with LC_{50} values ranging from 2.78 to 6.86 ppm. Two *S. littoralis* populations demonstrated the low levels of resistance at resistance ratios (RR) between 17.9- and 22.3-fold and three populations showed moderate resistance at 33.5 to 42.8-fold compared with a susceptible population. The findings demonstrated that diethyl maleate (DEM) and piperonyl butoxide (PBO) have considerable synergistic effects on the toxicity of emamectin benzoate.

Conclusion: The results suggest that emamectin benzoate resistance in the *S. littoralis* populations studied may be caused by the enzyme systems monooxygenase (MO) and glutathione S-transferase (GST).

Highlights

- Emamectin benzoate efficacy is decreasing in populations of *Spodoptera littoralis*.
- Emamectin benzoate resistance in *S. littoralis* field populations ranges from low to moderate.
- The field populations' monooxygenase and glutathione S-transferase activity were higher than those of the susceptible population.

Keywords: Carboxylesterase, Emamectin benzoate resistance, Glutathione S-transferases, Monooxygenase, Synergism.

1. Introduction

Chemicals are an effective strategy for managing the cotton leafworm, *Spodoptera littoralis* (Boisduval, 1833) (Lepidoptera: Noctuidae), one of the most destructive pests in Egypt and other nations. However, this pest is highly capable of developing insecticide resistance [1,2]. According to the Arthropod Pesticide Resistance Database, there more than 100 reported cases of resistance of *S. littoralis*. One of the new chemical pesticides utilized successfully in the management of cotton leafworm resistance is emamectin benzoate. However, it has been shown that various pests, particularly Lepidoptera species, are resistant to emamectin benzoate [3,4].

A naturally occurring substance called avermectin that is obtained from the soil bacterium *Streptomyces avermitilis* (Streptomycetaceae) is the basis of the semi-synthetic bioinsecticide known as emamectin benzoate [5]. In the insect nervous system, emamectin benzoate acts as a permanent activator of the chloride channel. The insect feeding process and muscular activity are suppressed by this chemical which ultimately results in insect death [6]. It is registered to control numerous lepidopteran pests in vegetables and field crops at low use rates in a number of countries, including Egypt, because of its broad-spectrum of activity and high degree of efficacy [6,7].

Enzymatic response, an adaptive process in living things, has led to the development of insecticide resistance. The chemical stress can cause the synthesis of detoxifying enzymes such esterases (EST), glutathione S-transferases (GSTs), and cytochrome P450 monooxygenases (P450s) [8,9]. Moreover, the enhanced activity of these detoxification processes can confer cross-resistance to insecticides with the same mode of action or even to those with other modes of action [10,11,12]. Thus, studies on the resistance mechanisms may provide useful information for combating pest resistance and improving control efficacy [13].

There are no data on the state and mechanism of emamectin benzoate resistance in Egypt, despite evidence of resistance in lepidopterous pests in China [12,13], Iran [14], Pakistan [15], Brazil [16], and India [17]. To determine the current state of emamectin benzoate resistance in field populations of *S. littoralis* collected from five Egyptian Governorates, this study was carried out to evaluate the detoxifying enzymes and to identify any probable processes underlying *S. littoralis*'s emamectin benzoate resistance.

2. Materials and Methods

2.1. Collection and rearing of *Spodoptera littoralis*

Field populations of *Spodoptera littoralis* (Boisd.) were collected from cotton (*Gossypium hirsutum* L.) fields located in five Egyptian Governorates of El-Behera, El-Kalyobiya, El-Minufiya, Asyut, and Bani Sweif. The populations had been exposed to frequent application of various insecticides including emamectin benzoate in these fields. The egg masses of *S. littoralis* collected from different locations were brought to the laboratory and reared separated into glass jars (500 ml).

The susceptible laboratory population of *S. littoralis* was obtained from Department of Insect Population Toxicology, Central Agricultural Pesticides Laboratory, Agriculture Research Center, Giza, Egypt, that was reared without contact with any insecticides for more than 15 years.

Larvae from susceptible and field populations were reared on castor bean leaves (*Ricinus communis* L.) at 25 ± 1°*C*, 65 ± 5% relative humidity, and a 16-h light/8-h dark photoperiod. The third-

instar larvae of susceptible and field populations were used for bioassays and biochemical experiments (Figure 1).

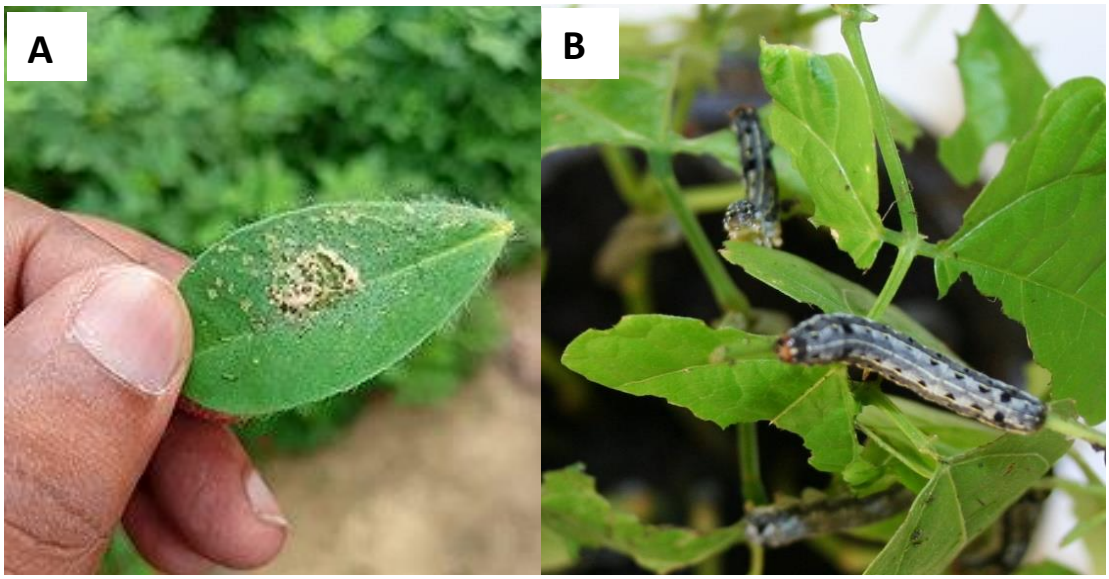

Figure 1. The stages of *Spodoptera littoralis* used in the experiment: (A) Egg masses and (B) Larvae.

2.2. Larvae bioassay

Bioassays were performed on one-day old third instar larvae of *S. littoralis* using the leaf dip method. Five concentrations of emamectin benzoate [Proclaim 5 SG®-Syngenta chemical] were diluted in water. Concentrations were prepared by making stock solutions, and then serial dilutions were made. The fresh castor bean leaves were cut to leaf discs (5 cm diameter). The leaf discs were dipped in each concentration in a glass beaker (250 ml capacity) for 10 seconds with gentle agitation. These exposed leaves were air dried at room temperature for 2 to 3 hours and they were kept in glass jars (500 ml). The leaf discs of control dipped in distilled water. The excess of insecticidal fluid present on the leaf disc was allowed to drain off and the discs were air-dried for 20 min. Third-instar larvae of *S littoralis* were released into jars to feed on the treated leaf discs. Each concentration was duplicated three times, with ten larvae (n=10) in each replication, a total of 180 larvae, including the control for each bioassay. Each jar was covered by two layers of tissue paper and glass cover fixed with rubber band to prevent larvae from escaping. Then, jars were placed in a breeding chamber at 25 ± 1 $°C$, 65 ± 5% relative humidity and a 16-h light/8-h dark photoperiod. The data of mortality was recorded after 48 hours of exposure to emamectin benzoate. The larvae were considered dead if they were unable to move after a gentle prodding with a fine brush.

2.3. Synergism assays

To measure the inhibition effect of detoxifying enzymes on emamectin benzoate resistance, three synergists piperonyl butoxide (PBO), triphenyl phosphate (TPP), and diethyl maleate (DEM) were used in combination with the insecticide. Five populations of El-Behera, El-Kalyobiya, El-Minufiya, Asyut, and Bani Sweif as well as susceptible population (as a reference population) of emamectin benzoate resistance were tested.

A constant concentration of each synergist (maximum concentration of

synergist, 10 mg L^{-1}) showed less than 10% mortality in the susceptible strain. The test method was similar to the above described leaf-dip bioassay. After pre-exposure of either PBO, TPP, or DEM, third-instar larvae of *S. littoralis* were transferred to emamectin benzoate-treated leaf discs.

2.4. Estimation of detoxification enzyme activity

Three principal detoxification enzymes involved in metabolic resistance, viz. carboxylesterase (CarE), glutathione S-transferase (GST), and monooxygenase (MO) activities were quantified. Fifty larvae of *S. littoralis* from two strains susceptible and field were homogenized on ice in 1000 mL of 0.1 M sodium phosphate buffer, pH 7.0, containing 1 mM EDTA, 1 mM dithiothreitol (DTT), 1 mM phenylthiourea, 1 mM PMSF, and 20% glycerol. The homogenates were then centrifuged at 4 °*C*, 15,000 rpm for 20 min. The supernatant obtained was used as a crude enzyme extract for spectrophotometric determination of the enzyme activities. Protein estimation was done following the Bradford's method [22], taking BSA as the standard. Samples were prepared in three replicates and OD was measured at 595 nm.

2.4.1. Total carboxylesterase (CarE) activity

Total CarE activity was measured by using α-naphthyl acetate (α-NA) as substrate [18]. The reaction mixture, contained 50 µL of α-NA (0.2 mM) and 50 µL of diluted enzyme solution in sodium phosphate buffer (0.2 M, pH 7.2). The mixture was then incubated at 37 °*C* for 15 min, the reaction was stopped by the addition 50 µL of the colorimetric reagent Fast Blue Stain (1% fast blue B salt in ethanol [w/v]). The absorbance was measured at 600 nm for the α-NA hydrolysis. The mean levels of the total cited esterase activity were based on protein content and α-NA standard curves. The α-esterase-specific activities were reported as µmoles of α-NA formed min^{-1} mg^{-1} protein.

2.4.2. Glutathione S-transferase (GST) activity

Glutathione S-transferase (GST) activity was assessed using 1-chloro2, 4-dinitrobenzene (CDNB) as a substrate through a previously described method Habig *et al.* [19]. Briefly, 3 mL of the reaction mixture consisted of 50 µL of 50 mM CDNB substrate solution, 150 µL of 50 mM GSH, and 30 µL of the diluted enzyme solution in sodium phosphate buffer (0.1 M, pH 7.5). The absorbance was measured using an ultraviolet spectrophotometer (Shimadzu UV-1201) at 340 nm with a read interval of 5 min. The GST specific activity was expressed as nmols min^{-1} mg^{-1} protein.

2.4.3. Monooxygenase (MO) activity

Monooxygenase (MO) activity was determined by 7-ethoxycoumarin (7-EC) as the substrate using a previously described method Ullrich and Weber (1972) [20] with some modifications by Van Pottelberge *et al.* [21]. The reaction mixture consisted of 50 µL of the diluted enzyme solution in sodium phosphate buffer (0.1 M, pH 7.5) containing 1 mM EDTA, 0.4 mM 7-EC in methanol, and 1 mM NADPH) in each well of a FLUOstar® Omega multi-mode microplate reader (BMG Labtech Ltd, Aylesbury, United Kingdom). The plate was incubated for 30 min at 30 °C in the dark while being gently shaken. To oxidize NADPH, 100 mM of GSSG in distilled water and 0.1 unit/µL of glutathione reductase were added to each well at 37 °C for 15 min. The reaction was stopped with 100 µL of 50% (v/v) acetonitrile in 50 mM tris/HCl buffer (pH 10). The fluorescence of 7-EC was measured at 460 nm while exciting it at

360 nm. The MO activity (7-EC-O-deethylation) was determined based on the 7-EC standard curve 45 to convert the initial velocity into the activity. The MO activity was expressed as nmols of 7-hydroxycoumarin formed/min/mg protein.

2.5. Statistical analysis

The mortality data were corrected using Abbott's formula [23]. Median lethal concentration (LC_{50}) values were calculated by probit analysis [24] using statistical software SPSS (version 19.0, SPSS Inc., Chicago, IL, USA). The resistance ratio (RR) was estimated as RR = LC_{50} (field population)/LC_{50} (the susceptible population). The level of emamectin benzoate resistance was described using RR as reported by Keiding (1980) [25]. Synergism ratio (SR) was calculated as SR = LC_{50} (emamectin benzoate without synergist)/LC_{50} (emamectin benzoate + synergist). Mean (± standard error, SE) enzyme activities recorded in larvae from the field populations were compared with those from larvae in the susceptible population.

3. Results

3.1. Insecticide resistance

Emamectin benzoate bioassay results for the different populations are presented in Table 1. In this study, using the leaf-dip method, the El-Behera population with an LC_{50} value of 3.57 ppm and the Asyut with an LC_{50} value of 6.86 ppm presented the lowest and the highest LC_{50} values, respectively. The significant difference existed in the LC_{50} of emamectin benzoate between reference (the susceptible population) and field populations. All field populations showed low to moderate levels of resistance to emamectin benzoate (Resistance Ratios (RR) from 17.9 to 42.9). The moderate resistance to emamectin benzoate was found in Bani Sweif, El-Kalyobiya, and Asyut. In the two populations (El-Behera and El-Minufiya), resistance ratio for emamectin benzoate were lower, ranging from 17.9 to 22.3.

Table 1. Susceptibility values (LC_{50}) and resistance ratios to emamectin benzoate in *Spodoptera littoralis* populations in Egypt

Population	LC_{50} (95% CL) [a] ppm	Slope (SE)	χ^2 (*df*) [b]	*p*-value	RR (fold) [c]
Susceptible	0.16 (0.13–0.20)	2.08(0.25)	1.93(3)	0.86	
Asyut	6.86 (3.28–10.52)	1.23(0.23)	0.30(3)	0.42	42.88
El-Behera	2.87 (1.98–3.38)	1.46(0.22)	3.26(3)	0.43	17.94
Bani Sweif	5.36 (4.06–6.66)	1.01(0.11)	1.40(3)	0.90	33.50
El-Minufiya	3.57 (2.94–4.39)	2.57(0.32)	1.57(2)	0.38	22.31
El-Kalyobiya	5.94 (2.72–7.47)	1.07(0.10)	1.97(3)	0.40	37.13

[a]95% confidence limits, [b] Chi-square value (χ^2), degrees of freedom (*df*), and *p*-value as calculated by probit analysis with SPSS. [c] RR (Resistance Ratio) = LC_{50} (field population)/LC_{50} (susceptible population).

3.2. Synergist bioassays

The estimated LC_{50} for *S. littoralis* from five field populations exposed to emamectin benzoate alone or with synergists (PBO, TPP, or DEM), are shown in Table (2). The significant differences were found in the estimated LC_{50} for the reference population (susceptible population) in emamectin benzoate-PBO, emamectin benzoate-TPP, or emamectin benzoate-DEM treatments. DEM and PBO exhibited a significant synergism on emamectin benzoate in the Asyut population (2.22- and 2.57-fold, respectively), and a very low synergistic

effect on emamectin benzoate in the El-Behera population (1.54- and 1.22-fold, respectively). However, TPP did not show any synergistic effect on emamectin benzoate in all field populations, as depicted in Figure 2.

Table 2. Susceptibility of emamectin benzoate-resistant *Spodoptera littoralis* exposed to emamectin benzoate alone or in the synergist presence

Population	Synergist	LC_{50} (95% CL) ppm	Slope (SE)	SR [a]
Susceptible	Emamectin benzoate	0.16 (0.13–0.20)	2.08(0.25)	
	+PBO	0.087 (0.038–0.12)	0.71(0.21)	0.54
	+DEM	0.061 (0.04–0.11)	0.36(0. 23)	0.38
	+TPP	0.021 (0.016–0.04)	0.74(0.11)	0.13
Asyut	Emamectin benzoate	6.86 (3.28–10.52)	1.23(0.23)	
	+PBO	2.67 (1.83–4.08)	4.41(0.75)	2.57
	+DEM	3.53 (2.94–4.39)	2.57(0.32)	1.94
	+TPP	6.64 (5.21–5.87)	1.54(0.95)	1.03
El-Behera	Emamectin benzoate	2.87 (1.98–3.38)	1.46(0.22)	
	+PBO	1.86 (0.92–2.13)	2.23(0.30)	1.54
	+DEM	2.35 (1.59–2.42)	2.24(0.23)	1.22
	+TPP	2.86 (2.26–3.48)	2.89(0.27)	1.00
Bani Sweif	Emamectin benzoate	5.36 (4.06–6.66)	1.01(0.11)	
	+PBO	2.67 (1.79–3.58)	1.45(0.22)	2.01
	+DEM	3.63 (2.67–4.69)	1.68(0.23)	1.48
	+TPP	5.28 (3.43–5.62)	2.67(0.39)	1.02
El-Minufiya	Emamectin benzoate	3.57 (2.94–4.39)	2.57(0.32)	
	+PBO	2.22 (0.13–0.20)	1.07(0.10)	1.60
	+DEM	2.57 (1.79–3.58)	1.45(0.22)	1.39
	+TPP	3.52 (2.09–4.52)	2.62(0.30)	1.01
El-Kalyobiya	Emamectin benzoate	5.94 (2.72–7.47)	1.07(0.10)	
	+PBO	2.68 (2.06–5.28)	1.23(0.22)	2.22
	+DEM	3.56 (3.14–6.23)	1.29(0.21)	1.67
	+TPP	5.75 (3.88–6.82)	2.45(0.37)	1.03

[a] SR (Synergistic Ratio) = LC_{50} (emamectin benzoate without synergist)/LC_{50} (emamectin benzoate + synergist).

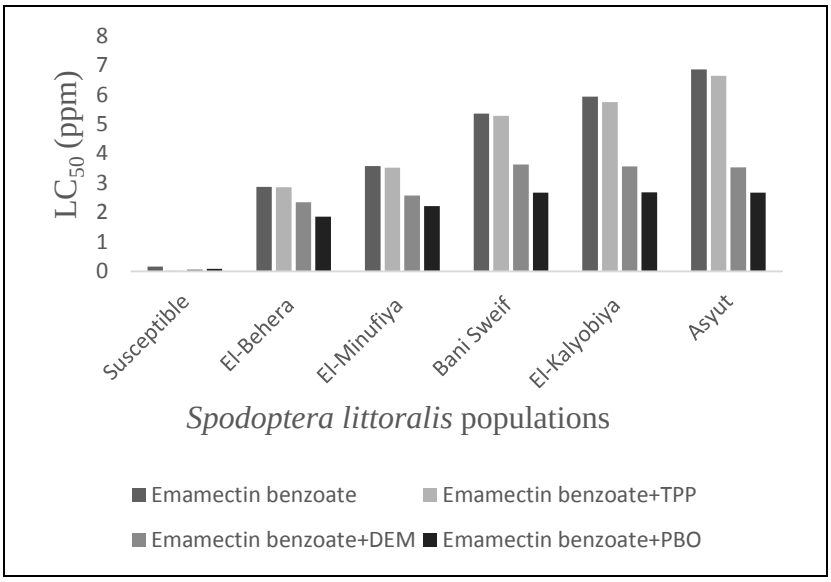

Figure 2. Susceptibility of emamectin benzoate-resistant *Spodoptera littoralis* exposed to emamectin benzoate alone or in the synergist presence.

3.3. Activity of detoxifying enzymes

Carboxylesterase (CarE), glutathione S-transferase (GST), and monooxygenase (MO) activities were evaluated in both the susceptible and field populations to provide further evidence on the involvement of enhanced metabolic mechanisms in emamectin benzoate resistance. The activities of CarE, GST, and MO were differed significantly among the field-collected *S. littoralis* populations and susceptible population (Table 3). The comparison of the MO average activity showed a significant difference between all field populations. Asyut population had the highest activity (4.19 pmol mg^{-1} min^{-1}) of MO. There was also a significant difference in GST between all tested field populations and susceptible population. The populations of Asyut and El-Behera showed the highest and lowest GST activity, respectively. The lowest determined activity was observed for the detoxifying enzyme CarE. These results were consistent with those from the synergism study.

Table 3. Metabolic enzyme activity in the susceptible and field populations of *Spodoptera littoralis* in Egypt

Population	GST activity [a] (nmol/min/mg protein)	[b]Ratio	[c] MO activity (pmol/min/mg protein)	[b]Ratio	[d] CarE activity (µmol/min/mg protein)	[b]Ratio
Susceptible	5.41 (±0.51) a		1.95 (±0.02) a		0.42 (±0.07) a	
Asyut	10.05 (±0.91) f	1.86	4.19 (±0.02) d	2.15	0.50 (±0.02) c	1.19
El-Behera	6.40 (±0.39) b	1.18	2.61 (±0.02) ab	134	0.43 (±0.04) a	1.02
Bani Sweif	8.03 (±0.61) d	1.48	3.34 (±0.02) bc	1.71	0.46 (±0.08) ab	1.10
El-Minufiya	6.96 (±0.34) bc	1.29	2.95 (±0.02) ab	1.51	0.44 (±0.03) a	1.05
El-Kalyobiya	9.11 (±0.33) e	1.68	4.01 (±0.02) d	2.06	0.48 (±0.09) ab	1.14

[a]CDNB was used as a substrate for the quantification of glutathione S-transferase activity. [b]Ratio= Activity of any field population/susceptible activity. [c]7-EC was used as a substrate for the quantification of monooxygenase activity. [d]Carboxylesterase detected against the substrate α-naphthyl acetate (α-NA). Using Tukey's test ($P \leq 0.05$), matched letters indicate no significant difference.

4. Discussion

A clear variability in emamectin benzoate susceptibility was detected between the field populations of *Spodoptera littoralis* (Boisd.), indicating that low to moderate levels of emamectin benzoate resistance occurred in the field populations. Emamectin benzoate

resistance in lepidopteran pests was reported by Zaka *et al.*, Ishtiaq *et al.,* and Muraro *et al.* [3,15,23].

To identify possible mechanisms involved in the varying susceptibility to emamectin benzoate among the tested field populations, the detoxification enzyme activities of monooxygenase (MO), glutathione S-transferase (GST), and carboxylesterases (CarE) were determined. The synergistic studies indicated that piperonyl butoxide (PBO) and diethyl maleate (DEM) increase the toxicity of emamectin benzoate in all field populations compared with the susceptible population [27]. Moreover, biochemical determination revealed that MO and GST activities are higher than in the susceptible population. Whereas, CarE is less involved in the degradation of emamectin benzoate in the resistant Egyptian population. An association was observed between the activities of the detoxification enzymes and the toxicity of emamectin benzoate, indicating that these enzymes may be a tolerance cause of the field population observed in this study. Stumpf and Nouen (2002), and Riga *et al.* (2014) [28,29] linked elevated levels of P450 and GST activity to abamectin resistance in strains of *Tetranychus urticae* (Acari: Tetranychidae) collected in the Netherlands, Brazil, and Colombia. Likewise, Siqueira *et al.* [27] reported that cytochrome P450 and GST activity are involved in abamectin resistance, but GST appears to be of the secondary importance as the mechanism of abamectin resistance in *Tuta absoluta* (Merck) resistant populations. The GST involvement in abamectin resistance was supported by another study in *Tetranychus urticae* (Acari: Tetranychidae) [30]. No correlation was observed between the CarE activity and resistance to emamectin benzoate in *Plutella xylostella* (Linnaeus) [31].

5. Conclusion

Low to moderate resistance to emamectin benzoate was found in the field populations tested in this study. Tests and synergistic measurements of the activity of detoxifying enzymes indicated a role for monooxygenase (MO) and glutathione S-transferase (GST) in the development of *Spodoptera littoralis* resistance to emamectin benzoate. Therefore, further investigations of the physiological mechanisms are needed to develop an appropriate strategy to prevent or delay the development of higher levels of emamectin benzoate resistance.

Abbreviation

CarE: carboxylesterase
GST: glutathione S-transferase
MO: monooxygenase

Authors' contributions

S.M.I. subject selection, study design, carried out the experiments, paper writing, collecting, interpretation of the data, and performing statistical analysis. The author read and approved the final manuscript.

Consent for publications

The author agrees to have read the manuscript and authorize the publication of the final version of the manuscript.

Conflict declaration

The authors declare that there is no conflict.

Conflict of interest:

None of the authors have any conflict of interest to declare.

Availability of data and material

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ORCID

Seham Mansour Ismail: https://www.orcid.org/0000-0002-4885-7383

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