

Current insecticide–resistance status and activity of detoxifying enzymes in field populations of *Bemisia tabaci* from Egypt

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Abstract

Little information is available about monitoring resistance to several insecticide classes in field populations of the whitefly, *Bemisia tabaci* (Gennadius) (Homoptera: Aleyrodidae), in Egypt. Thus, organophosphates chlorpyrifos-methyl, profenofos and primiphos-methyl, carbamates methomyl and carbosulfan, and pyrethroids α -cypermethrin, λ -cyhalothrin and deltamethrin were tested against *B. tabaci* adults collected from cotton fields in different Egyptian Governorates. Insecticide-treated adults were compared with a susceptible laboratory strain for determining the resistance ratio (RR) and quantifying the activity of the detoxifying enzymes acetylcholinesterase (AChE), carboxylesterase (CarE), glutathione S-transferase (GST) and monooxygenases (MOs). Higher resistance was developed in field populations collected from Faiyum and Beheira Governorates against chlorpyrifos-methyl, with RR₅₀ and RR₉₀ of 19.8, 47.0 and 204.0, 28.0, respectively, and against λ -cyhalothrin, with RR₅₀ and RR₉₀ of 28.5, 17.2 and 25.0 and 15.6, respectively. Generally, *B. tabaci* collected from Minya Governorate displayed the lowest resistance level. Also, among the tested insecticides, λ -cyhalothrin induced, in general, the highest resistance level. There was a correlation between the relative increase in the resistance of λ -cyhalothrin-resistant *B. tabaci* populations and the relative increase in MOs and/or GST activity. This study provides baseline information on the insecticide resistance status and detoxifying enzymes activity of whitefly populations in cotton fields in Egypt and for monitoring future insecticide resistance development..

Keywords: Conventional insecticides, resistance, *Bemisia tabaci*, acetylcholinesterase, carboxylesterase, glutathione S-transferase, monooxygenases

1. Introduction

The cotton whitefly, *Bemisia tabaci* (Gennadius) (Homoptera: Aleyrodidae), is a pest with great agricultural importance worldwide. During the past few years, it has become an increasingly important pest of cotton and vegetables in Egypt and also worldwide (El-Kady and Devine 2003). This may be attributed to the random use of insecticides and the subsequent selection of insecticide resistance (Denholm *et al.* 1998), changes in agronomic practices (Dittrich *et al.* 1986) and the immigration of previously known whitefly biotypes (Abd-Rabu 1999). *B. tabaci* not only feeds on leaves resulting in delayed growth and even death of the plants, but also deposits honeydew on leaves which often lead to sooty mold and reduction in photosynthesis (Zhang

et al. 2012). Additionally, it is also known to transmit various plant viruses (Jones 2003).

The most challenge in management of *B. tabaci* is its ability to develop insecticide resistance to virtually every chemical that has never been used against it (Denholm *et al.* 1998). During the past 25 years, *B. tabaci* control in Egypt has been dependent exclusively on conventional insecticides such as organophosphates (OPs), carbamates and pyrethroids, and by the late 1980s, at least some of these broad-spectrum insecticides have been failed as a control measure against this insect pest (Abdallah 1991; Sobiha *et al.* 1997). Nevertheless, monitoring resistance to several insecticide classes in field populations of *B. tabaci* in Egypt has been undertaken by a few researchers (El-

Kady and Devine 2003; Farghaly 2010; Farghaly *et al.* 2010, 2014). The most common reasons for insect resistance to most insecticides are enhanced metabolic detoxification and target-site insensitivity (Wilson and Ashok 1998; Siegfried and Zera 1994; Zhang *et al.* 2015).

Thus, the present study aimed to (1) monitor resistance in field populations of *B. tabaci* collected from different Egyptian Governorates to OPs chlorpyrifos-methyl, profenofos and primiphos-methyl, carbamates methomyl and carbosulfan, and pyrethroids α -cypermethrin, λ -cyhalothrin and deltamethrin compared to a susceptible laboratory strain, and (2) quantify the activity of the detoxifying enzymes acetylcholinesterase (AChE), carboxylesterase (CarE), glutathione S-transferase (GST) and monooxygenases (MOs) in *B. tabaci* adults. This study may be helpful in developing and implementing a resistance management program against *B. tabaci* in cotton fields in Egypt

2. Material and Methods

2.1 Whiteflies

Field strains of *B. tabaci* adults were collected, using a mouth aspirator, in the early morning hours from cotton fields in different localities in seven Governorates (Menoufia, Gharbia, Faiyum, Beheira, Minya, Dakahlia and Qalyubia) covering its whole distribution in Egypt (Table 1, Fig. 1) (Farghaly *et al.* 2014). The collected samples were transported to the laboratory in a cool box and identified following the taxonomic key described by Martin *et al.* (2000). The susceptible strain collected on cotton plant (*Gossypium barbadense* L.) from Minya Governorate was reared without exposure to insecticides for the past 10 years. This strain was used as a standard for evaluating the magnitude of resistance in field collected strains. All insects were maintained on cotton plant (*G. barbadense* L.) in screen cages (50 × 35 × 35 cm) at 26 ± 2°C, 50–60% RH and 16: 8 h (L: D) photoperiod as described by Coudriet *et al.* (1985).

2.2 Insecticides

Eight commercial insecticide formulations were used: chlorpyrifos-methyl (EC 50%), profenofos (EC

72%) and primiphos-methyl (EC 50%) (OPs); methomyl (WP 90%) and carbosulfan (WP 25%) (carbamates); α -cypermethrin (EC 15%), λ -cyhalothrin (EC 20%) and deltamethrin (EC 2.5%) (pyrethroids). These insecticides were regularly used in cotton fields in Egypt against *B. tabaci*.

2.3 Bioassay protocol

The collected whitefly adults were tested within 2 h of arrival in the laboratory. Bioassay methods for obtaining concentration–mortality response lines were followed using a leaf–dip method according to Feng *et al.* (2010) with some modifications. The insecticides were dissolved and diluted with distilled water containing Triton X-100 (0.1%), as non-ionic wetting agent, to obtain the desired concentrations (100 mL each). Cotton leaf discs (55 mm diameter) were dipped for 10 sec in the insecticide solution and were air dried. Treated leaves were placed with their adaxial surface downwards on a bed of thin layer of agar (2%) in a small Petri dish (55 mm diameter) placed in a small cage. Control groups were performed using cotton leaf discs dipped in distilled water containing

Table 1. Collection sites of *Bemisia tabaci* strains from different Egyptian Governorates.

Strain/Governorate (collection site)		°N	°E
Menoufia (Berket Ashmoun)	El-Sabaa,	30.52 (30.63, 30.29)	30.99 (31.08, 31.03)
Gharbia (Zefta, Kotoor)		30.88 (30.67, 30.97)	31.06 (31.23, 30.95)
Faiyum (Sinnuris, Atsa, Ibshway)		29.30 (29.41, 29.25, 29.39)	30.84 (30.86, 30.69, 30.63)
Beheira (Etay El-Barud, Wadi El-Natron)		30.61 (30.90, 30.43)	30.43 (30.68, 30.24)
Minya (El-Idwa, Mallawi)	Samalut,	28.11 (28.69, 28.29, 27.70)	30.11 (30.76, 30.70, 30.80)
Dakahlia (MitGhamr, Bilqas)		31.03 (30.71, 31.23)	31.23 (31.25, 31.36)
Qalyubia (Qalyoub, Benha)		30.41 (30.19, 30.46)	31.21 (31.20, 31.18)

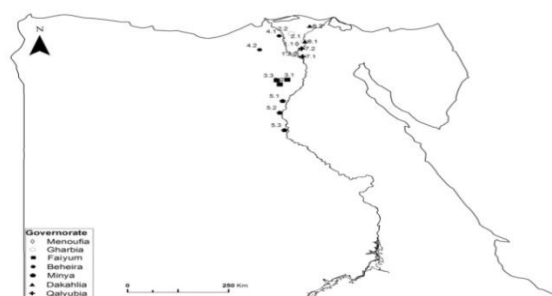


Fig. 1. Map of Egypt showing the collection sites of *Bemisia tabaci* in the different Governorates. 1.1: Berket El-Sabaa; 1.2: Ashmoun (Menoufia Governorate); 2.1: Zefta; 2.2: Kotoor (Gharbia Governorate); 3.1: Sinnuris; 3.2: Atsa; 3.3: Ibshway (Faiyum Governorate); 4.1: Etay El-Barud; 4.2: Wadi El-Natrun (Beheira Governorate); 5.1: El-Idwa; 5.2: Samalut; 5.3: Mallawi (Minya Governorate); 6.1: MitGhamr; 6.2: Bilqas (Dakahlia Governorate); 7.1: Qalyoub; 7.2: Benha (Qalyubia Governorate).

Triton X-100 only. Twenty adults were placed onto each leaf disc. Seven serial concentrations of each insecticide were tested. Each insecticide concentration was repeated five times. The dishes were inverted for the insects to orientate normally and confined in a large controlled room at $26 \pm 2^\circ\text{C}$, 50–60% RH and 16: 8 h (L: D) photoperiod. Handling mortality was estimated within 1 h. Mortality was recorded after 48h. Resistance ratio (RR50 or RR90) of the collected strains was obtained by dividing their calculated LC50 or LC90 values, respectively, by that of the susceptible laboratory strain (reference population). The following criteria proposed by **Mazarri and Georgiou (1995)** were adopted to classify the resistance level of populations: low ($\text{RR} < 5$), moderate ($5 < \text{RR} < 10$) or high ($\text{RR} > 10$).

2.4 Detoxifying enzymes assay

2.4.1 Sample preparation

Since it was not known which insecticide the whiteflies collected from the field had developed resistance against it, λ -cyhalothrin was selected where bioassays in this study revealed that the highest RR in *B. tabaci* adults treated with this insecticide had been developed. *B. tabaci* adults collected from the above mentioned seven Governorates were treated with the LC50 of λ -cyhalothrin using a leaf-dip method as described above. The survivors were homogenized separately in distilled water (50 mg mL⁻¹) using Teflon-glass homogenizer surrounded with a jacket of crushed ice. Each homogenate was then centrifuged at 7656 g force for 15 min at -4°C using cooling centrifuge. The resultant supernatants (enzyme and protein source) were labeled and stored at -20°C till use for further

determination of AChE, CarE, GST and MFO activity, as well as total protein content compared to the susceptible laboratory strain. Each assay was repeated three times.

2.4.2 AChE assay

AChE activity was measured according to the methods described by **Simpson et al. (1964)** using acetylcholine bromide (AChBr) as a substrate. The reaction mixture contained 200 μL of the enzyme solution, 0.5 mL phosphate buffer (0.067 M, pH 7) and 0.5 mL AChBr (3 mM). The test tubes were incubated at 37°C for exactly 30 min. One mL of alkaline hydroxylamine (equal volume of 2M hydroxylamine chloride and 3.5M NaOH) and 0.5 mL HCl (one part concentrated HCl: two parts distilled water) were added to the test tubes. The mixture was shaken thoroughly and allowed to stand for 2 min. Then, 0.5 mL ferric chloride solution (0.9M) dissolved in HCl (0.1M) was added and mixed well. The decrease in AChBr resulting from hydrolysis by AChE was read at 515 nm using double beam UV/visible spectrophotometer (Milton Roy Spectronic 1201 UV-Visible, USA).

2.4.3 CarE assay

CarE activity was measured following the same methods used for measuring AChE activity (**Simpson et al. 1964**), except the use of methyl n butyrate (MeB) as a substrate. The decrease in MeB resulting from hydrolysis by CarE was read spectrophotometrically at 515 nm.

2.4.4 GST assay

GST activity was determined based on the technique of **Habig *et al.* (1974)** using 1-chloro-2,4-dinitrobenzene (2,4-CDNB) as a substrate. The reaction mixture comprised of 10 μ L reduced glutathione (GSH) (10 mM) in sodium phosphate buffer (100 mM, pH 6.5) and 10 μ L of the enzyme solution. The reaction was initiated by adding 10 μ L of 2,4-CDNB (6 mM in methanol) resulting in a final volume of 30 μ L. The plates were immediately transferred to absorbance microplate reader (BioTek Instruments, Inc., Winooski, VT, USA). The reactions were allowed to continue for 5 min and absorbance readings were taken at 340 nm automatically once per min against blanks (wells containing all reaction components except the enzyme solution). The increase in absorbance was linear throughout the 5 min reading interval. An extinction coefficient of 9.6 mM⁻¹cm⁻¹ was used to calculate the amount of 2,4-CDNB conjugated.

2.4.5 MOs assay

MOs activity was detected through the transformation of p-nitroanisole to p-nitrophenol through O-demethylation via the enzyme p-nitroanisole-O-demethylase based on the methods of **Hansen and Hodgson (1971)** with slight modifications. The standard incubation mixture contained 1 mL sodium phosphate buffer (0.1 M, pH 7.6), 1.5 mL enzyme solution, 0.2 mL NADPH (final concentration 1 mM), 0.2 mL glucose-6-phosphate (final concentration 1 mM) and 50 μ g glucose-6-phosphate dehydrogenase. The reaction was initiated by the addition of p-nitroanisole in 10 μ L acetone to give a final concentration of 0.8 mM and was incubated for 30 min at 37°C. The incubation period was terminated by the addition of 1 mL HCl (1N), and p-nitrophenol was extracted with CHCl₃ and NaOH (0.5 N). The absorbance of NaOH solution was measured at 405 nm. An extinction coefficient of 14.28 mM⁻¹cm⁻¹ was used to calculate the concentration of 4-nitrophenol.

2.5 Total protein

Total protein content was determined according to **Bradford (1976)**.

2.6 Statistical analysis

The percentage mortality of treated larvae was corrected against that of the control using Abbott's formula (**Abbott, 1925**). Then, the corrected mortality was subjected to Probit analysis (**Finney, 1971**). Data of the biochemical assays were analyzed using one-way analysis of variance (ANOVA). When the ANOVA statistics were significant ($p < 0.05$), the means were compared by Duncan's multiple range test. All the analyses were computed by IBM® SPSS® Statistics 21.0 (IBM Corp., Armonk, NY, USA).

3. Results

3.1. Resistance studies

3.1.1. OPs resistance

Based on the RR50, *B. tabaci* field populations developed resistance ranged from low to high to chlorpyrifos-methyl and primiphos-methyl. However, low to moderate resistance was obtained against profenofos (Table 2). High resistance level (RR50 and RR90) was developed to chlorpyrifos-methyl in *B. tabaci* populations collected from Faiyum and Beheira Governorates and primiphos-methyl in samples collected from Beheira Governorate. Moderate resistance was developed to chlorpyrifos-methyl (Dakahlia and Qalyubia Governorates), profenofos (Faiyum and Qalyubia Governorates) and primiphos-methyl (Faiyum and Qalyubia Governorates). Low resistance was recorded to chlorpyrifos-methyl (Menoufia, Gharbia and Minya Governorates), profenofos (Menoufia, Gharbia, Beheira, Dakahlia Governorates) and primiphos-methyl (Menoufia, Gharbia, Minya and Dakahlia Governorates). In general, *B. tabaci* displayed the highest resistance to chlorpyrifos-methyl in the majority of the regions of cotton cultivation in Egypt (Table 2).

Table 2. Resistance of *Bemisia tabaci* strains collected from different Egyptian Governorates to the organophosphates chlorpyrifos-methyl, profenofos and primiphos-methyl, compared to the susceptible laboratory strain.

Strain	Chlorpyrifos-methyl					Profenofos					Primiphos-methyl				
	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ± SE	χ ² (df)	RR ₅₀ , RR ₉₀	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ± SE	χ ² (df)	RR ₅₀ , RR ₉₀	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ± SE	χ ² (df)	RR ₅₀ , RR ₉₀
Suscep.	82.5, 297.9	61.2–108.3 211.3–495.7	2.3 ±0.31	1.6 (5)	– –	87.7, 217.2	68.6–112.5, 160.3–358.5	3.2 ±0.5	1.1 (5)	– –	24.1, 177.3	13.02–36.4, 109.9–403.2	1.5 ±0.3	4.3 (5)	– –
Me	158.0, 1084.3	97.2–221.0, 670.9–2721.2	1.5 ±0.28	1.01 (5)	1.9, 3.6	107.0, 521.7	81.4–137.6, 372.9–835.2	1.9 ±0.20	8.3 (5)	1.2, 2.4	35.6, 335.6	32.4–46.2, 182.6–5396.8	1.3 ±0.23	2.8 (5)	1.5, 1.9
G	294.0, 2393.2	198.9–412.7, 1424.3–5774.7	1.4 ±0.21	3.71 (5)	3.6, 8.0	89.8, 640.1	63.2–121.5, 419.5–1213.2	1.5 ±0.19	3.7 (5)	1.0, 2.9	119.0, 751.5	79.6–200.2, 498.7–1450.0	1.6 ±0.23	4.6 (5)	4.9, 4.2
F	1631.9, 14011.7	1100.2–3524.7, 5343.2–27118.3	1.4 ±0.36	1.1 (5)	19.8, 47.0	623.3, 5768.0	450.9–909.0, 3195.0–9351.9	1.3 ±0.16	3.8 (5)	7.1, 26.6	212.3, 3464.3	132.1–325.4, 1566.3–7404.8	1.1 ±0.19	2.5 (5)	8.8, 19.5
B	1679.9, 8421.0	1219.6–2857.2, 4257.8–12135.7	1.8 ±0.40	2.1 (5)	20.4, 28.3	195.1, 2024.8	138.3–270.2, 1232.7–4328.8	1.3 ±0.14	10.3 (5)	2.2, 9.3	303.2, 1706.3	217.4–408.6, 1069.3–3965.3	1.7 ±0.28	7.7 (5)	12.6, 9.6
Mi	129.4, 656.9	85.3–182.8, 412.9–1442.9	1.4 ±0.22	1.23 (5)	1.6, 2.2	299.2, 536.8	212.4–409.3, 355.8–1352.1	1.6 ±0.27	2.6 (5)	3.4, 2.5	59.6, 363.8	43.8–79.0, 243.7–657.3	1.6 ±0.19	3.9 (5)	2.5, 2.1
D	723.4, 5113.2	522.3–1421.16, 2894.3–13281.6	1.5 ±0.33	8.5 (5)	8.8, 17.2	242.1, 3637.5	100.0–422.2, 1392.6–8696.9	1.1 ±0.31	2.1 (5)	2.8, 16.7	108.1, 1094.0	63.7–157.2, 681.9–1628.4	1.1 ±0.23	1.2 (5)	4.5, 6.2
Q	594.2, 4246.5	415.6–841.8, 2370.7–8328.5	1.5 ±0.27	1.5 (5)	7.2, 14.3	598.3, 3622.6	439.6–894.4, 1966.7–6786.9	1.6 ±0.16	1.8 (5)	6.8, 16.7	189.0, 1685.1	135.7–263.5, 990.9–3878.3	1.3 ±0.17	6.4 (5)	7.8, 9.5

RR: Resistance ratio = LC50 or LC90 of field strain/LC50 or LC90 of susceptible strain; 95% CL: 95% Confidence limit; B: Beheira Governorate; D: Dakahlia Governorate; F: Faiyum Governorate; G: Gharbia Governorate; Me: Menoufia Governorate; Mi: Minya Governorate; Q: Qalyubia Governorate

3.1.2 Carbamates resistance

Resistance, based on the RR50, of *B. tabaci* to methomyl was moderate in samples collected from Faiyum Governorate, while it was low in samples collected from the other Governorates. Resistance to carbosulfan was high in samples collected from Gharbia Governorate. However, it was moderate in samples collected from the other Governorates, except in Minya and Qalyubia Governorates where the level of resistance was low (Table 3).

3.1.3 Pyrethroids resistance

B. tabaci collected from the seven Egyptian Governorates exhibited low to moderate resistance to α -cypermethrin, and low to high resistance to λ -cyhalothrin and deltamethrin, based on the RR50 (Table

4). High resistance was developed to λ -cyhalothrin in samples collected from Faiyum, Beheira and Dakahlia (RR50 and RR90) and Gharbia (RR50) Governorates, and to deltamethrin (RR50) in samples collected from Faiyum and Beheira Governorates. Moderate resistance was recorded to α -cypermethrin (Faiyum and Beheira Governorates) and deltamethrin (Gharbia, Dakahlia and Qalyubia Governorates). However, low resistance was recorded to α -cypermethrin (Menoufia, Gharbia, Minya, Dakahlia and Qalyubia Governorates), λ -cyhalothrin (Menoufia, Minya and Qalyubia Governorates) and deltamethrin (Menoufia and Minya Governorates) (Table 4). In general, among the eight tested insecticides, λ -cyhalothrin induced the highest resistance to all the collected strains of *B. tabaci* (Tables 2–4).

Table 3.Resistance of *Bemisia tabaci* collected from the different Egyptian Governorates to the carbamates methomyl and carbosulfan, compared to the susceptible laboratory strain.

Strain	Methomyl					Carbosulfan				
	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ± SE	χ^2 (df)	RR ₅₀ , RR ₉₀	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ± SE	χ^2 (df)	RR ₅₀ , RR ₉₀
Suscep.	401.7, 1470.1	192.2–765.2, 844.16–3963.3	1.2±0.68	2.08 (5)	–, –	6.3, 169.9	2.5–10.3, 73.9–217.7	0.9±0.27	2.5 (5)	– –
Me	811.5, 2864.8	496.1–1248.3, 1494.3–4223.6	2.0±0.39	1.36 (5)	2.0, 1.9	39.0, 338.5	23.8–56.0, 187.0–1165.4	1.4±0.27	3.1 (5)	6.2, 2.0
G	974.5, 6107.7	698.4–1458.2, 2767.3–53287.4	1.1±0.23	4.65 (5)	2.4, 4.2	64.8, 325.6	48.4–87.3, 205.4–732.7	1.8±0.29	2.3 (5)	10.3, 1.9
F	2212.2, 13596.1	1571.0–3959.1, 6323.6–19581.0	1.6±0.37	1.02 (5)	5.5, 9.2	53.9, 303.9	39.3–70.37, 205.9–535.4	1.1±0.22	1.9 (5)	8.6, 1.8
B	1862.9, 9114.7	1299.5–3220.1, 36749.9–11370.0	1.5±0.37	3.06 (5)	4.6, 6.2	57.7, 230.8	44.3–74.6, 158.0–425.8	2.1±0.31	2.8 (5)	9.2, 1.4
Mi	612.1, 2646.2	239.5–994.0, 1450.8–5673.1	1.4±0.56	2.06 (5)	1.5, 1.8	17.1, 202.7	9.4–25.6, 114.3–591.3	1.2±0.21	4.7 (5)	2.7, 1.2
D	920.4, 6155.1	528.1–1327.2, 2783.3–8211.2	1.8±0.29	1.72 (5)	2.3, 4.2	46.8, 225.5	32.2–63.8, 143.7–533.9	1.9±0.34	6.6 (5)	7.4, 1.3
Q	822.8, 5281.2	485.2–1158.9, 3019.7–11004.2	1.6±0.37	1.07 (5)	2.0, 3.6	24.2, 424.8	14.6–33.5, 184.8–644.3	1.8±0.31	3.4 (5)	3.8, 2.5

RR: Resistance ratio = LC₅₀ or LC₉₀ of field strain/LC₅₀ or LC₉₀ of susceptible strain; 95% CL: 95% Confidence limit; B: Beheira Governorate; D: Dakahlia Governorate; F: Faiyum Governorate; G: Gharbia Governorate; Me: Menoufia Governorate; Mi: Minya Governorate; Q: Qalyubia Governorate

3.2 Detoxifying enzymes activity

3.2.1 AChE activity

Table 5 shows that AChE activity of *B. tabaci* collected from Minya Governorate was significantly increased ($p < 0.05$) due to treatment with the LC50 of λ -cyhalothrin compared to the susceptible laboratory strain. Whereas, field populations collected from Menoufia, Faiyum and Qalyubia Governorates showed a significant decline ($p < 0.05$) in AChE activity. The change in AChE activity of the other Governorates was insignificant.

3.2.2 CarE activity

Treatment of *B. tabaci* with the LC50 of λ -cyhalothrin revealed a significant decrease ($p < 0.05$) in

CarE activity of the samples collected from Menoufia, Gharbia, Faiyum, Minya and Qalyubia Governorates compared to the susceptible laboratory strain. Whilst no significant change in CarE activity was observed in samples collected from Beheira and Dakahlia Governorates (Table 5).

3.2.3 GST activity

B. tabaci collected from Beheira, Minya, Dakahlia and Qalyubia Governorates showed a significant increase ($p < 0.05$) in GST activity compared to the susceptible laboratory strain due to the stress of λ -cyhalothrin. On the contrary, GST

Table 4. Resistance of *Bemisia tabaci* collected from the different Egyptian Governorates to the pyrethroids α -cypermethrin, λ -cyhalothrin and deltamethrin, compared to the susceptible laboratory strain.

Strain	α -Cypermethrin					λ -Cyhalothrin					Deltamethrin				
	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ±SE	χ^2 (df)	RR ₅₀ , RR ₉₀	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ±SE	χ^2 (df)	RR ₅₀ , RR ₉₀	LC ₅₀ , LC ₉₀ (ppm)	95% CL (LC ₅₀ , LC ₉₀)	Slope ±SE	χ^2 (df)	RR ₅₀ , RR ₉₀
Suscep.	12.0, 105.0	6.2–18.8, 52.2–322.1	1.4 ±0.33	2.4 (5)	– –	4.3, 24.1	2.9–5.8, 15.4–52.9	1.7 ±0.27	3.3 (5)	– –	9.4, 53.3	3.4–15.5, 38.2–90.3	1.7 ±0.74	2.6 (5)	– –
Me	30.8, 144.3	22.9–40.6, 95.6–291.7	1.9 ±0.29	3.2 (5)	2.6, 1.4	7.2, 28.4	5.4–9.3, 20.8–45.7	2.2 ±0.29	2.5 (5)	1.7, 1.2	29.3, 69.7	23.7–35.3, 54.8–103.3	3.4 ±0.53	2.1 (5)	3.1, 1.3
G	40.2, 123.9	31.1–49.8, 93.3–195.9	2.6 ±0.4	1.7 (5)	3.4, 1.2	51.8, 258.8	38.7–69.3, 164.7–469.1	1.8 ±0.29	3.1 (5)	12.0, 10.7	89.3, 360.8	67.8–118.8, 232.1–660.1	2.1 ±0.38	1.5 (5)	9.5, 6.8
F	71.1, 270.3	55.2–91.1, 189.1–482.4	2.2 ±0.31	2.7 (5)	5.9, 2.6	122.7, 413.9	94.1–156.62, 290.6–787.4	2.4 ±0.41	1.1 (5)	28.5, 17.2	138.1, 491.7	107.2–192.7, 309.4–935.9	2.3 ±0.42	2.4 (5)	14.7, 9.2
B	96.1, 326.1	75.5–126.9, 220.4–646.7	2.4 ±0.31	7.1 (5)	8.0, 3.1	107.5, 375.8	84.5–146.4, 242.2–579.6	2.4 ±0.41	1.2 (5)	25.0, 15.6	105.2, 322.9	81.0–172.0, 189.6–681.9	2.6 ±0.65	1.5 (5)	11.2, 6.1
Mi	23.9, 169.6	17.2–35.0, 90.8–387.0	1.5 ±0.27	3.2 (5)	2.0, 1.6	12.3, 39.4	8.9–14.2, 28.5–65.2	2.4 ±0.31	1.1 (5)	2.9, 1.6	15.9, 56.4	11.8–20.3, 41.2–93.4	2.3 ±0.35	2.0 (5)	1.7, 1.1
D	59.7, 136.2	49.8–72.1, 106.2–203.8	3.8 ±0.52	1.7 (5)	5.0, 1.3	52.9, 254.4	33.0–73.9, 166.9–550.2	1.9 ±0.35	4.4 (5)	12.3, 10.6	63.4, 372.9	46.2–87.3, 222.7–965.6	1.7 ±0.28	2.4 (5)	6.7, 7.0
Q	23.2, 151.1	16.8–33.4, 83.9–370.5	1.6 ±0.27	1.7 (5)	1.9, 1.4	10.9, 49.7	8.2–14.1, 34.8–85.4	1.84 ±0.25	1.8 (5)	2.5, 2.1	54.4, 187.2	42.7–71.9, 124.7–402.3	2.4 ±0.41	2.61 (5)	5.8, 3.5

RR: Resistance ratio = LC₅₀ or LC₉₀ of field strain/LC₅₀ or LC₉₀ of susceptible strain; 95% CL: 95% Confidence limit; B: Beheira Governorate; D: Dakahlia Governorate; F: Faiyum Governorate; G: Gharbia Governorate; Me: Menoufia Governorate; Mi: Minya Governorate; Q: Qalyubia Governorate

Activity of *B. tabaci* collected from Gharbia and Faiyum Governorates was significantly declined ($p < 0.05$) compared to the susceptible laboratory strain. The

3.2.4 MOs activity

Exposure of *B. tabaci* collected from Beheira, Minya and Dakahlia Governorates to the LC₅₀ of λ -cyhalothrin resulted in a significant increase ($p < 0.05$) in MOs activity compared to the susceptible laboratory strain. By contrast, MOs activity of the strains collected from Menoufia, Gharbia, and Faiyum Governorates was significantly decreased ($p < 0.05$). The change in MOs activity of the populations collected from Qalyubia Governorate was insignificant (Table 5).

Change in GST activity of Menoufia strain was insignificant (Table 5).

4. Discussion

B. tabaci has developed high resistance to some commonly used insecticides (Ahmad 2007; Bacci *et al.* 2007; Roditakis *et al.* 2009; Farghaly 2010; Caballero *et al.* 2013; Avicor *et al.* 2014; Farghaly *et al.* 2014). Abdallah (1991) found that low levels of resistance to primiphos-methyl and profenofos in Egyptian whiteflies were noted in 1989/1990.

Table 5. Activity (mean $\pm$ SD) of acetylcholinesterase (AChE), carboxylesterase (CarE), glutathione S-transferase (GST) and mixed-function oxidases (MOs) of *Bemisia tabaci* strains collected from different Egyptian Governorates and treated with the LC50 of λ -cyhalothrin of the respective strain compared to the susceptible laboratory strain.

Strain	AChE ($\mu\text{g AChBr/min/g protein}$)	CarE ($\mu\text{g MeB} \times 10^3/\text{min/g protein}$)	GST (nmol 2,4- CDBN Congugated/min/g rotein)	MOs (nmol pNP produced/ min/g protein)
Susceptible	0.11 $\pm$ 0.003a	12.83 $\pm$ 0.502a	22.06 $\pm$ 0.740a	1.11 $\pm$ 0.007a
Menoufia	0.06 $\pm$ 0.004b	1.26 $\pm$ 0.115b	20.63 $\pm$ 0.547a	0.93 $\pm$ 0.052b
Gharbia	0.13 $\pm$ 0.009a	1.42 $\pm$ 0.136b	18.19 $\pm$ 0.588b	0.73 $\pm$ 0.010c
Faiyum	0.09 $\pm$ 0.004c	8.39 $\pm$ 0.282c	17.53 $\pm$ 0.600b	0.80 $\pm$ 0.012bc
Beheira	0.13 $\pm$ 0.009a	13.80 $\pm$ 0.411a	28.08 $\pm$ 0.305c	1.53 $\pm$ 0.037d
Minya	0.20 $\pm$ 0.004d	8.96 $\pm$ 0.240c	28.32 $\pm$ 0.495c	1.45 $\pm$ 0.028d
Dakahlia	0.13 $\pm$ 0.012a	13.03 $\pm$ 0.126a	29.00 $\pm$ 0.387c	1.56 $\pm$ 0.042d
Qalyubia	0.08 $\pm$ 0.005c	2.21 $\pm$ 0.202d	29.25 $\pm$ 0.733c	1.14 $\pm$ 0.013a

AChE: Acetylcholinesterase; CDBN: 1-Chloro-2,4-dinitrobenzene; MeB: Methyl *n* butyrate; pNP: *p*-Nitrophenol. Means followed by different letters in the same column are significantly different ($p < 0.05$) using one-way analysis of variance (ANOVA), followed by Duncan's multiple range test for comparison among the means.

Some years later, *B. tabaci* collected from cotton fields were found to be 2– to 5–fold more resistant to primiphos-methyl and profenofos than a laboratory standard (**Refaie and Ayad 1998**). In comparison, our results indicated that *B. tabaci* collected from cotton in Egypt was 1.47– to 12.57–fold more resistant to primiphos-methyl, and 1.02– to 7.10–fold more resistant to profenofos than the susceptible laboratory strain. **El-Kady and Devine (2003)** reported that Egypt is considered one of the largest consumers of pyrethroids worldwide, and most of them were used on cotton, with 10 to 12 applications per season were common in the cotton crop. They also reported that carbamates are widely used to control *B. tabaci* in Egypt, and in 1994 this class of insecticides accounted for 31% of the total quantity of insecticides used on cotton in this country. Therefore, it was expected that a considerable resistance against pyrethroids and carbamates would be present. **Farghaly (2010)** pointed out that resistance of four field strains of *B. tabaci* in Egypt to pyrethroids was higher than that to OPs and carbamates during the seasons 2006 and 2007. In comparison to our results, **Avicor et al. (2014)** found that the LC50 of *B. tabaci* collected

from field vegetables in Ghana to λ -cyhalothrin, using a leaf dip technique, was 0.12-0.55 mL-1.

The higher resistance developed by *B. tabaci* to chlorpyrifos-methyl, primiphos-methyl, carbosulfan and λ -cyhalothrin and deltamethrin in samples collected from Faiyum and Beheira Governorates might make the control of *B. tabaci* in these Governorates is a major problem. Accordingly, it is not recommended to use these insecticides in these Governorates. On the contrary, the application of the tested insecticides in the current investigation in Minya Governorate would not impair field performance against *B. tabaci*, where the lower level of resistance was developed. The lower resistance developed by whiteflies collected from Minya Governorate (an Upper Egypt Governorate) compared to other Governorates (Lower Egypt Governorates) may be due to lower agricultural development (pesticide application) paid by the Egyptian government to Upper Egypt Governorates compared to that paid for Lower Egypt Governorates. Though insecticide resistance to λ -cyhalothrin has been reported in *B. tabaci* populations in

this study, this insecticide continues to be effective in reducing populations of some homopteran species (Salem *et al.* 2009) including *B. tabaci* (Li *et al.* 2009, Salem *et al.* 2009).

Since the susceptibility status of *B. tabaci* to insecticides could alter from year to year (Ahmad *et al.* 2010), monitoring of their resistance status to the insecticides tested in the current study and other insecticides should be sustained whilst farmers are encouraged on the judicious use of insecticides.

Typically, enhanced metabolic detoxification among insects may contribute to insecticide resistance (Low *et al.* 2007), the metabolic detoxification system in insects consists of three major groups of enzymes. The phase I detoxification enzymes, acting on a broad range of substrates directly to reduce their toxicity, are represented by cytochrome P450. The phase II enzymes, including GST, UDP-glucuronosyltransferases (UGTs), and CarE, facilitate the excretion of hydrophobic toxic compounds by improving their hydrophilicity. Several studies indicated that increased levels of detoxification gene expression are known to result in increased levels of detoxifying enzymes that are responsible for insecticide resistance (Karunker *et al.* 2008; Liu *et al.* 2011; Schuler, 2011; Gong *et al.* 2013; Zhang *et al.* 2015).

Pyrethroid resistance in field populations of *B. tabaci* is primarily a consequence of the overproduction of esterase isozymes that metabolize and sequester pyrethroid insecticides (Young *et al.* 2006). B-type esterases hydrolyze pyrethroids and their enhanced activity confers resistance in whiteflies (Byrne *et al.* 2000). Resistance to pyrethroids in whiteflies has also been associated with mutations in the para sodium channel gene (Morin *et al.* 2002; Farghaly *et al.* 2010) and L925I and T929V, VGSC gene (Gauthier *et al.* 2014).

AChE is one of the most conserved enzymes of higher eukaryotes (Russell *et al.* 2004). It is widely known for its role in hydrolyzing acetylcholine, but it also has another much less studied role in the development, mutation and maintenance of vertebrate and invertebrate nervous system (Grisaru *et al.* 1999; Ranson *et al.* 2002). The F331W mutation in the AChE enzyme ace1 gene associated with resistance to OPs was

fixed in *B. tabaci* (Vassiliou *et al.* 2011; Gauthier *et al.* 2014). The increased activity of AChE may arise from a quantitatively increased production of the enzyme (Fournier *et al.* 1992) or a qualitatively modified enzyme with a higher catalytic efficiency (Walsh *et al.* 2001; Vontas *et al.* 2002a; Weill *et al.* 2003).

GST played a major role in the detoxification of profenofos in *B. tabaci* (Kandil *et al.* 2008). This might be attributed to conjugation of lipid peroxidation products (Vontas *et al.* 2002b). However, several authors did not find a significant increase in GST activity of insecticide-resistant *B. tabaci* populations (Rauch and Nauen 2004; Zhang *et al.* 2012).

The present study showed that there was a correlation between the relative increase in the resistance of λ -cyhalothrin-resistant *B. tabaci* populations and the relative increase in MOs and/or GST activity. In agreement with this finding, several authors found that the major mechanism in *B. tabaci* appeared to be enhanced detoxification by cytochrome P450 MOs (Rauch and Nauen 2003; Karunker *et al.* 2008). Kandil *et al.* (2008) found that piperonyl butoxide synergized profenofos toxicity in resistant strains of whiteflies. Moreover, they also elucidated the role of diethylmaleate as inhibitor for GST in profenofos-resistant whiteflies.

In conclusion, of the seven Governorates studied, it appears that moderate to high resistance in *B. tabaci* populations against the studied insecticides had been occurred in Gharbia, Faiyum, Beheira, Dakahlia and Qalyubia Governorates (~57% of the regions of cotton cultivation in Egypt), making resistance is a national problem. Development of resistance depends in part on the occurrence of "refugia" for susceptible populations (Georghiou 1994) and on gene flow among populations colonizing different habitats in a given area. Studies on the genetic structure of the *B. tabaci* population together with the identification of resistance genes will allow a better understanding of the evolution of resistance. The development of a resistance management scheme is being considered through the area-wide implementation of integrated pest management. This study provides baseline information on the insecticide resistance status and detoxifying enzymes activity of whitefly populations in cotton fields in Egypt and for monitoring future insecticide resistance development

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References

- Abbott WJ, 1925.** A method of computing the effectiveness of an insecticide. *J Econ Entomol*, 18, 265–267.
- Abdallah MD, 1991.** A general view of the resistance problem of cotton pests in Egypt. *Resist Pest Manag*, 3, 22–25.
- Abd-Rabu S, 1999.** New records of whiteflies in Egypt. *Egyptian J Agric Res*, 77, 1143–1146.
- Ahmad M, Arif MI, Naveed M, 2010.** Dynamics of resistance to organophosphate and carbamate insecticides in the cotton whitefly *Bemisia tabaci* (Hemiptera: Aleyrodidae) from Pakistan. *J Pestic Sci*, 83, 409–420.
- Ahmad M., 2007.** Potentiation/antagonism of pyrethroids with organophosphate insecticides in *Bemisia tabaci* (Homoptera: Aleyrodidae). *J Econ Entomol*, 100, 886–893.
- Avicor SW, Eziah VY, Owusu EO, Wajidi MFF, 2014.** Insecticide susceptibility of *Bemisia tabaci* to Karate® and Cydim Super® and its associated carboxylesterase Activity. *Sains Malays*, 43, 31–36.
- Bacci L, Crespo ALB, Galvan TL, Pereira EJG, Picanço MC, Silva GA, Chediak M, 2007.** Toxicity of insecticides to the sweetpotato whitefly (Hemiptera: Aleyrodidae) and its natural enemies. *Pest Manag Sci*, 63, 699–706.
- Bale HJ, 1981.** Insecticide Resistance—A Practical Assessment. *Bull Entomol Soc Am*, 27, 261–262.
- Bradford MM, 1976.** A rapid and sensitive method for the quantification of microgram quantities of proteins utilizing the principle of protein–dye binding. *Analyt Biochem*, 72, 248–254.
- Byrne FJ, Gorman KJ, Cahill M, Denholm I, Devonshire AL, 2000.** The role of B-esterases in conferring insecticide resistance in the tobacco whitefly, *Bemisia tabaci* (Genn.). *Pest Manag Sci*, 56, 867–874.
- Caballero D, Cyman S, Schuster DJ, 2013.** Baseline susceptibility of *Bemisia tabaci*, biotype b (Hemiptera: Aleyrodidae) to chlorantraniliprole in southern Florida. *Fla Entomol*, 96, 1002–1008.
- Coudriet DL, Prabhaker N, Kishaba N, Meyerdirk DE, 1985.** Variation in the developmental rate on different hosts and overwintering of the sweet potato whitefly *Bemisia tabaci* (Homoptera: Aleyrodidae). *Environ Entomol*, 14, 516–519.
- Denholm I, Cahill M, Dennehy TJ, Horowitz AR, 1998.** Challenges with managing insecticide resistance in agricultural pests exemplified by the whitefly, *Bemisia tabaci*. *Philosoph Transact Roy Soci London (Series B, Biol Sci)*, 353, 1757–1767.
- Dittrich V, Hassan SO, Ernst GH, 1986.** Development of a new primary pest of cotton in the Sudan: *Bemisia tabaci*, the whitefly. *Agric Ecosyst Environ*, 17, 137–142.
- El-Kady H, Devine GJ, 2003.** Insecticide resistance in Egyptian populations of the cotton whitefly, *Bemisia tabaci* (Hemiptera: Aleyrodidae). *Pest Manag Sci*, 59, 865–871.
- Farghaly SF, 2010.** Biochemical monitoring for insecticide resistance in whitefly *Bemisia*

- tabaci*. Am-Euras J Agric Environ Sci, 8, 383–389.
- Farghaly SF, Abou-Yousef HM, Singab M, Ghoneim YF, 2010.** Identification of mutation in the *Bemisia tabaci* (Genn.) para sodium channel gene associated with resistance to pyrethroids. Egypt J Agric Res, 88, 153–166.
- Farghaly SF, Hamama HM, Dawood AE, 2014.** Role of cytochrome P450 gene in insecticide susceptibility of the whitefly, *Bemisia tabaci* (Homoptera, Aleyrodidae) in Egyptian Governorates. Inter J Biol Sci Appl, 1, 62–71.
- Feng YT, Wu QJ, Wang SL, Chang XL, Xie W, Xu BY, Zhang YJ, 2010.** Cross-resistance study and biochemical mechanisms of thiamethoxam resistance in B-biotype *Bemisia tabaci* (Homoptera: Aleyrodidae). Pest Manag Sci, 66, 313–318.
- Finney DJ, 1971.** Probit Analysis. 3rd edition. Cambridge University Press, Cambridge.
- Fournier D, Bride JM, Hoffman F, Karch F, 1992.** Acetylcholinesterase: two types of modifications confer resistance to insecticides. J Biol Chem, 267, 14270–14274.
- Gauthier N, Clouet C, Perrakis A, Kapantaidaki D, Peterschmitt M, Tsagkarakou A, 2014.** Genetic structure of *Bemisia tabaci* Med populations from home-range countries, inferred by nuclear and cytoplasmic markers: impact on the distribution of the insecticide resistance genes. Pest Manag Sci, 70, 1477–1491.
- Georghiou GP, 1994.** Principles of insecticide resistance management. Phytoprotection, 75, 51–59.
- Gong Y, Li T, Zhang L, Gao X, Liu N, 2013.** Permethrin induction of multiple cytochrome P450 genes in insecticide resistant mosquitoes, *Culex quinquefasciatus*. Inter J Biol Sci, 9, 863–871.
- Grisaru D, Sternfeld M, Eldor A, Glick D, Soreq H, 1999.** Structural roles of acetylcholinesterase variations in biology and pathology. Eur J Biochem, 264, 672–686.
- Habig WH, Pabst MJ, Jakoby WB, 1974.** Glutathione S-transferase. The first enzymatic step in mercapturic acid formation. J Biol Chem, 249, 7130–7139.
- Hansen IG, Hodgson E, 1971.** Biochemical characteristics of insect microsomes: N- and O-demethylation. Biochem Pharmacol, 20, 1569–1578.
- Jones DR, 2003.** Plant viruses transmitted by whiteflies. Eur J Plant Pathol, 109, 195–219.
- Kandil MA, Saleh AY, El-Dieb WH, Farghaly SF, 2008.** Resistance mechanisms of whitefly *Bemisia tabaci* (Homoptera: Aleyrodidae) to thiamethoxam and profenofos. Asian J Biol Sci, 1, 33–38.
- Karunker I, Benting J, Lueke B, Ponge T, Nauen R, Roditakis E, Vontas J, Gorman K, Denholm I, Morin S, 2008.** Overexpression of cytochrome P450 CYP6CM1 is associated with high resistance to imidacloprid in the B and Q biotype of *Bemisia tabaci* (Homoptera: Aleyrodidae). Insect Biochem Mol Biol, 38, 634–644.
- Li L, Xue M, Ren GW, Li QL, Zhang QC, Wang HT, 2009.** Toxicity of several insecticides to different instars nymphs of *Bemisia tabaci* and the repellent actions to the adults. Acta Phytophyl Sin, 36, 359–365.
- Liu N, Li T, Reid WR, Yang T, Zhang L, 2011.** Multiple cytochrome P450 genes: Their constitutive overexpression and permethrin induction in insecticide resistant mosquitoes, *Culex quinquefasciatus*. Plos One, 6, e23403.

- Low WY, Ng HL, Morton CJ, Parker MW, Batterham P, Robin C, 2007.** Molecular evolution of glutathione S-transferases in the genus *Drosophila*. *Genetics*, 177, 1363-1375.
- Martin JH, Mifsud D, Rapisarda C, 2000.** The whiteflies (Homoptera: Aleyrodidae) of Europe and Mediterranean Basin. *Bull Entomol Res*, 90, 407–448.
- Mazarri MB, Georgiou GP, 1995.** Characterization of resistance to organophosphate, carbamate and pyrethroid insecticides in field populations of *Aedes aegypti* from Venezuela. *J Am Mosq Control Assoc*, 11, 315–322.
- Morin S, Williamson MS, Goodson SJ, Brown JK, Tabashnik BE, Dennehy TJ, 2002.** Mutations in the *Bemisia tabaci* sodium channel gene associated with resistance to a pyrethroid plus organophosphate mixture. *Insect Biochem Mol Biol*, 32, 1781-1791.
- Ranson H, Claudianos C, Ortel F, Abgrall C, Hemingway J, Sharakhova MV, Unger M.F, Collins FH, Feyereisen R, 2002.** Evaluations of supergene families associated with insecticide resistance. *Science*, 298, 179–181.
- Rauch N, Nauen R, 2003.** Biochemical markers linked to neonicotinoid cross-resistance in *Bemisia tabaci* (Homoptera: Aleyrodidae). *Arch Insect Biochem Mol Biol*, 54, 165–176.
- Rauch N, Nauen R, 2004.** Characterization and molecular cloning of a glutathione S-transferase from the whitefly *Bemisia tabaci* (Homoptera: Aleyrodidae). *Insect Biochem Mol Biol*, 34, 321–329.
- Refaie BM, Ayad FA, 1998.** Integument ultrastructure of *Bemisia tabaci* adults controlled by primiphos-methyl and profenofos (Homoptera: Aleyrodidae). *Bull Entomol Soc Egypt*, 76, 135–147.
- Roditakis E, Grispou M, Morou E, Kristoffersen JB, Roditakis N, Nauen R, Vontas J, Tsagkarakou A, 2009.** Current status of insecticide resistance in Q biotype *Bemisia tabaci* populations from Crete. *Pest Manag Sci*, 65, 313-322.
- Roush RT, Miller GL, 1986.** Considerations for design of insecticide resistance monitoring programs. *J Econ Entomol*, 79, 293–298.
- Russell RJ, Claudianos C, Campbell PM., Horne I, Sutherland TD, Oakeshott JG, 2004.** Two major classes of target site insensitivity mutations confer resistance to organophosphate and carbamate insecticides. *Pestic Biochem Physiol*, 79, 84–93.
- Salem HA, Sabry EKH, Aref NB, 2009.** Insecticide potential of buprofezin and lambda-cyhalothrin on mealybugs, aphids and whitefly infesting ornamental-medicinal plants. *Bull Ent Soc Egypt (Econ Ser)*, 35, 189-201.
- Schouest JR, Miller TA, 1988.** Factors influencing pyrethroid toxicity in pink bollworm (Lepidoptera: Gelechiidae) implications for resistance management. *J Econ Entomol*, 81, 431–436.
- Schuler MA, 2011.** Review P450s in plant–insect interactions. *Biochim Biophys Acta (BBA)-Proteins and Proteomics*, 1814, 36-45.
- Siegfried BA, Zera AJ, 1994.** Partial purification and characterization of a green bug (Homoptera: Aphididae) esterases associated with resistance to parathion. *Pestic Biochem Physiol*, 49, 132–137.
- Simpson DR, Bulland DL, Lindquist DA, 1964.** A semimicrotechnique for estimation of cholinesterase activity in boll weevils. *Ann Entomol Soc Amer*, 57, 367–371.
- Sobiha AK, Zidan ZH, Mowafy MM, Marzouk AS, 1997.** Effect of certain insecticide regimes on

some sucking insects and natural enemies inhabiting cotton plants. *Ann Agric Sci*, 42, 665–674.

Vassiliou V, Emmanouilidou MPA, Morou E, Vontas J, Tsagkarakou A, Roditakis E, 2011. Insecticide resistance in *Bemisia tabaci* from Cyprus. *Insect Sci*, 18, 30-39.

Vontas JG, Hejazi MJ, Hawkes NJ, Cosmidis N, Loukas M, Hemmingway J, 2002a. Resistance-associated point mutations of organophosphate insensitive acetylcholinesterase in the olive fruitfly, *Bactrocera oleae*. *Insect Mol Biol*, 11, 329–336.

Vontas JG, Small GJ, Nikou DC, Ranson H, Hemmingway J, 2002b. Purification, molecular cloning and heterologous expression of a glutathione S-transferase from the rice brown planthopper, *Nilaparvata lugens*. *Biochem J*, 362, 329–337.

Walsh SB, Dolden TA, Moores GD, Kristensen M, Lewis T, Devonshire AL, Williamson MS, 2001. Identification and characterization of mutations in housefly (*Musca domestica*) acetylcholinesterase involved in insecticide resistance. *Biochem J*, 359, 175–181.

Weill M, Lutfalla G, Mogenson K, Chandre F, Berthomieu A, Berticat C, Pasteur N, Philips A, Fort P, Raymond M, 2003. Insecticide resistance in mosquito vectors. *Nature*, 423, 136–137.

Wilson TG, Ashok M, 1998. Insecticide resistance resulting from an absence of target-site gene product. *Proceed Nation Acad Sci (USA)*, 95, 14040–14044.

Young SJ, Gunning RV, Moores GD, 2006. Effect of pretreatment with piperonyl butoxide on pyrethroid efficacy against insecticide-resistant *Helicoverpa armigera* (Lepidoptera: Noctuidae) and *Bemisia tabaci* (Sternorrhyncha: Aleyrodidae). *Pest Manag Sci*, 62, 114–119.

Zhang B, Kong F, Zeng X, 2015. Detoxification enzyme activity and gene expression in diafenthiuron resistant whitefly, *Bemisia tabaci*. *J Agric Sci*, 7, 66 – 76.

Zhang N, Liu C, Yang F, Dong S, Han Z, 2012. Resistance mechanisms to chlorpyrifos and f392W mutation frequencies in the acetylcholine esterase aceI allele of field populations of the tobacco whitefly, *Bemisia tabaci* in China. *J Insect Sci*, 12, 41.

الملخص العربي

رصد الوضع الحالي للمقاومة ونشاط إنزيمات إزالة السموم في آفة الذبابة البيضاء

في بعض محافظات مصر. *Bemisia tabaci* (Genn.)

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الملخص العربي

في مصر قليل من المعلومات المتاحة عن رصد المقاومة لعدة أنواع من المبيدات الحشرية في مجال مكافحة الذبابة البيضاء مثل مركبات البيروثرويدات منها α سايبير ميثرين- λ سياهاالوثرين و دلتا ميثرين و مجموعة المبيدات الفوسفورية منها كلوروبيريفوس- بروفينوفوس و بريميفوس ميثيل ، أخيرا مجموعة الكاربامات ومنها مبيدي الميثوميل و كاربوسولفان.

وقد تم تجميع آفة الذبابة البيضاء من حقول القطن في بعض محافظات مصر ، وقد تم أيضا مقارنة هذه العشائر بالسلالة المعملية الحساسة لتحديد معامل المقاومة و دراسة نشاط إنزيمات إزالة السمية مثل اسيتيل كولين استيريز ، كاربوكسي استيريز و جلوتاثيون-S- استيريز و مونوكسي جينيز .

وقد حددت أعلى مقاومة في عشيرتي محافظة الفيوم والبحيرة للمركب الفوسفوري كلوروبيريفوس ميثيل و تحديد قيمة معامل المقاومة القاتل ل ٥٠ % و ٩٠ % من العشيرة ١٩,٨ و ٤٧,٠ جزء في المليون علي التوالي لمحافظة الفيوم ، ٢٨,٠ و ٢٠٤,٠ جزء في المليون لمحافظة البحيرة علي التوالي.

وبصفة عامة وجد أن آفة الذبابة البيضاء المجمعة من محافظة المنيا أظهرت اقل مستوى من المقاومة ، وأيضا من بين كل المبيدات المختبرة وجد ان العشائر المختلفة قد أظهرت مستوى مقاومة عالية لمبيد λ سياهاالوثرين وقد وجد أيضا ان هناك علاقة بين الزيادة النسبية لمقاومة السلالة المقاومة لهذا المبيد والزيادة في النشاط الانزيمي ل GST و MOs .

هذه الدراسة تمدنا بمعلومات اساسية عن المقاومة للمبيدات ونشاط الانزيمات المزيلة للسمية في العشائر المختلفة الذبابة البيضاء في مصر .